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BRITISH JOURNAL OF ANAESTHESIA

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EDITORIAL

THE 'COMPLEAT' ANAESTHETIST

THE *Symposium on Pulmonary Ventilation** edited by Dr. Harbord and Professor Woolmer long expected has at last arrived. It is tastefully bound and clearly printed and we feel impelled to congratulate the publishers on its excellent appearance. We must leave to others the task of reviewing the contents, but if they in any way match the outside we hope this volume is the first of a long series which all interested in the subjects discussed will want to possess.

The appearance of the symposium draws attention to the enormous extension of the territory over which anaesthesia has spread itself in the last thirty years. The improved financial position of anaesthetists which has followed on the establishment of the Association of Anaesthetists and inauguration of the National Health Service has attracted men to the subject who, although they might have been interested in anaesthesia, would formerly have felt that they could not afford to take the subject up as their life work. This influx of talent has naturally led to a great increase in research and opened up so many fields to the anaesthetist of to-day that he is sometimes embarrassed with riches and may find it difficult to determine how best to spend his time, energy, and talents.

It must never be forgotten that the first duty of the anaesthetist is to anaesthetize his patient, to become a skilled technician, and a good doctor; many will be content to stop there. The problems arise in the operating theatre, the solution often is only to be found at the University Department in conjunction with a pharmacologist, a physiologist, and a biochemist. It adds immensely to the interest and life of an anaesthetist if he can find the time and has the ability to take part in such investigations. It is ridiculous that senior men 'Circled through all experience' should spend all their time doing routine work equally well done by their juniors. On the other hand many men when they become Professors and Directors of Departments seem no longer able to pursue directly the subject to which so far they have devoted their lives and must spend the rest of their days in sitting on committees. 'They also serve . . .' and in the long view, their contribution to the specialty may be greater. It seems unfortunate, however, that after years of research and teaching they may appear to become almost exclusively employed in political activity and administration. To steer between these unpleasant extremes needs foresight and judgement.

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CIRCULATORY CHANGES ACCOMPANYING RESPIRATORY ACIDOSIS DURING HALOTHANE (FLUOTHANE) ANAESTHESIA IN MAN*

BY

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THE haemodynamic effects of halothane differ considerably from those of the inhalational anaesthetic agents in common use. Data on the response of the circulation of human subjects to halothane is therefore of interest. In the present report the condition of cardiac rhythm, arterial blood pressure and heart rate during halothane anaesthesia at normal levels of alveolar carbon dioxide tension in fifteen subjects is described. No pre-anaesthetic medication was given to these individuals; steady states of anaesthesia were sought and all measurements were made prior to operation. In view of the importance which has been ascribed to hypercarbia in the initiation of cardiac arrhythmias and posthypercarbic hypotension, observations were also made during and after deliberate elevation of the inspired concentration of carbon dioxide. The results in most instances are compared and contrasted with those obtained during cyclopropane anaesthesia.

METHODS

The fifteen subjects studied were patients admitted for minor surgical operations. The ages of these subjects, of whom fourteen were female, ranged from 15 to 49 years. On clinical examination thirteen were found to be in good health, one had symptomless hypertension and one inactive pulmonary tuberculosis.

None of the patients was receiving any drug therapy and no pre-anaesthetic medication was given. On arrival in the anaesthetic room each subject was given 15 litres of oxygen per minute by face mask for 5–10 minutes from a Heidbrink anaesthetic machine. A one-way valve (Ruben) was included to make a nonrebreathing system. Halothane was then added from a Fluotec vaporizer and the concentration gradually increased until the induction of anaesthesia was complete, usually in 7–10 minutes. A metal oral airway was inserted in each subject. In most instances the induction of anaesthesia was quiet and satisfactory, although excitement was marked in two subjects and moderate in two others. The high inspired concentration of oxygen—over 95 per cent—the adequate pulmonary ventilation as judged by end-expired P_{CO_2} values and the pink colour of the mucous membranes and nailbeds of the subjects led us to believe that no subject was hypoxic at any time. Respiration was spontaneous and unassisted.

All observations were made before the operation was started. The average time of study was 3 hours, during which time 200–500 ml of 5 per cent glucose in water were given intravenously.

The inspired halothane concentration was analyzed by means of a thermal conductivity cell which had been calibrated with weighed amounts of halothane vaporized in oxygen. The percentages of halothane were recorded at 5-minute intervals. Oxygen from the Heidbrink machine was passed through the Fluotec vaporizer and the thermal conductivity cell into a 5-litre bag and then to the patient through the Ruben valve. Thermal conductivity readings were not taken when carbon dioxide was added to the inspired

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gases, for the low conductivity of the latter gas made it difficult to determine the concentration of halothane in the mixture. It is believed that carbon dioxide did not interfere with the functioning of the Fluotec.

The carbon dioxide tension in the expired gases was measured with a Liston-Becker infrared carbon dioxide analyzer using the microcatheter technique of Collier, Affeldt and Farr (1955). The gases were withdrawn by inserting a 20 gauge needle through the rubber face mask and directing it into the lumen of the metal oral airway. It was found that halothane in the concentrations used in this study did not interfere with infrared determination of carbon dioxide. End-expired values of P_{CO_2} were used throughout the studies.

Prior to the induction of anaesthesia the arterial pressure was measured by the auscultatory (Riva-Rocci) method in ten cases, and directly through a needle inserted in the brachial or femoral artery in the remainder. In nine individuals arterial puncture was successful shortly after the induction of anaesthesia, and direct measurements of pressure were therefore possible throughout the remaining period of study in fourteen of the fifteen individuals. In one subject arterial puncture failed, and blood pressure measurements were continued by the auscultatory method. When the direct method was used the pressure pulse was transduced by a strain gauge and the mean arterial pressure calculated by planimetric integration of the pressure curves.

The electrocardiogram (leads I and II) was recorded using needle electrodes. In five cases the needles were inserted while the subject was awake, and in others not until after induction of anaesthesia. The tracings were interpreted according to standard methods (Bellet, 1953).

The arterial blood pressure, the electrocardiogram and the concentration of carbon dioxide were continuously recorded on a Grass polygraph. A paper speed of 150 mm/min was used, with records being taken at 1500 mm/min at intervals of several minutes and during periods of special interest.

The inspired concentrations of halothane ranged from 0.4 to 2.5 per cent. The Fluotec setting was often 3 per cent, but thermal conductivity readings indicated a value consistently lower by 0.3–0.5 per cent. A steady concentration of anaesthetic was administered for at least 20 minutes in every instance in an effort to achieve stable states of anaesthesia.

Varying degrees of hypercarbia were then produced by adding carbon dioxide from a high pressure cylinder. The P_{CO_2} was elevated slowly until ventricular arrhythmias were produced, was maintained at this level for varying periods up to 25 minutes, and was then reduced.

It was planned to study the effect of two or more steady levels of anaesthesia in each subject. When P_{CO_2} was deliberately elevated during low inspired halothane concentrations (0.4–1 per cent) coughing and gagging on the airway made studies unsatisfactory. For this reason, in most subjects hypercarbia was first studied during higher concentrations of halothane. A return was then made to a normal P_{CO_2}. A stable state of anaesthesia at a lower concentration of halothane was sought and hypercarbia again produced. The "arrhythmia threshold" was found to be reproducible in the same subject when carbon dioxide inhalation was repeated under similar conditions, but the haemodynamic changes were not. For these reasons all data relevant to arrhythmia production will be included in this report, but only the haemodynamic measurements obtained during the first period of stable anaesthesia and the first episode of hypercarbia will be presented.

The aim in each study was to determine whether cardiac arrhythmias occurred during elevation of P_{CO_2} and, if so, at what level of end-expired P_{CO_2}. In some subjects the reproducibility of a "critical arrhythmia level" was studied. In others the influence of different concentrations of halothane and different rates of rise of P_{CO_2} upon the "arrhythmia threshold" was examined.

Standard statistical methods were used to determine the significance of differences between experimental observations (Moroney, 1956). A difference was considered significant when the p value was less than 0.05.

RESULTS

Cardiac Arrhythmias.

Normocarbia. When the end-expired P_{CO_2} lay within the normal range, cardiac rhythm was also normal, with minor exceptions. Depression of the

P wave without increase in the P–R interval was present at some time during anaesthesia in three subjects. In two other individuals slight depression of the S–T segment occurred briefly in one, and S–T elevation in the other.

The concentrations of halothane inspired ranged between 0.4 and 2.5 vols. per cent. When the respirations were unassisted, the end-expired P_{CO_2} was greater the higher the anaesthetic concentration ($p < 0.01$). The P_{CO_2} averaged 42 mm Hg (S.D. = 6) during periods when constant concentrations of halothane in oxygen were being inhaled.

In one subject soft tissue obstruction in the respiratory tract developed following the induction of anaesthesia, resulting in hypercarbia (P_{CO_2} approximately 60 mm Hg) and a burst of ventricular premature contractions which disappeared with restoration of a patent airway. Another individual gagged repeatedly on an oral airway during light anaesthesia and this was followed by a similar arrhythmia which also vanished when the respirations became regular.

Hypercarbia. Inhalation of carbon dioxide produced abnormalities of cardiac rhythm consisting of atrioventricular nodal rhythm, ventricular extrasystoles and multifocal ventricular tachycardia, in each of fifteen subjects, and in 36 of 38 trials. The ventricular arrhythmias appeared at a characteristic and reproducible P_{CO_2} "threshold" in each subject at similar concentrations of halothane and rates of rise of P_{CO_2}.* This is shown in table I. There was no consistent relation between the concentration of halothane respired and the P_{CO_2} threshold (fig. 1). The effect of changing the halothane concentration was studied in more detail in seven cases (who served as their own controls). Three were found to show a lesser susceptibility to arrhythmia production when halothane concentration was low than when it was high. The opposite was the case in the four others.

TABLE I

Reproducibility of P_{CO_2} Threshold for Cardiac Arrhythmias.

Subject No.	Per cent halothane	Rate of P_{CO_2} increase (mm Hg/min)	P_{CO_2} arrhythmia threshold (mm Hg)
1	1.9	2	66
	1.8	4	68
5	1.8	3	80
	1.8	4	80
9	1.1	4	116
	1.1	4	120
13	2.0	3	86
	2.0	2	82
14	1.0	5	117
	1.0	5	120

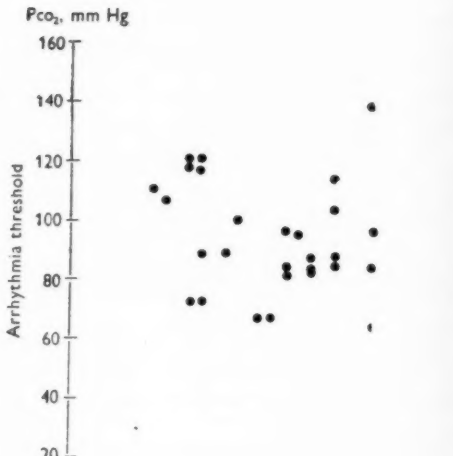

FIG. 1

P_{CO_2} arrhythmia threshold in relation to halothane concentration.

The average end-expired P_{CO_2} level at which ventricular arrhythmias first appeared was 92 mm Hg (range 60–140 mm Hg). Subjects who developed multifocal ventricular tachycardia did so at a significantly ($p < 0.05$) lower level (76 ± 16 (S.D.) mm Hg).

*In a similar study during cyclopropane anaesthesia Lurie and associates (1958) showed that the "arrhythmia threshold" was over-estimated in proportion to the rate of P_{CO_2} rise. A similar observation was made in the present study. For this reason only the results obtained when P_{CO_2} was elevated at less than 5 mm Hg/min have been included in the present report.

Ventricular extrasystoles were always preceded by normal sinus rhythm except for the minor changes in P wave and S–T segment already mentioned. Multifocal ventricular tachycardia developed in six instances in five subjects. In each case it was preceded by ventricular premature beats. The type of arrhythmia readily changed in a given subject without apparent reason although one type predominated during each period of hypercarbia. Elevating the P_{CO_2} 10–20 mm Hg above the arrhythmia "threshold" was without effect either upon the frequency of ventricular extrasystoles or the number of ectopic foci.

Atrioventricular nodal rhythm occurred on eight occasions in five subjects during hypercarbia. In three instances it was associated with retrograde conduction, and in one with a "wandering pacemaker". Nodal rhythm appeared, if it occurred at all, with the onset of ventricular extrasystoles. In three subjects it persisted after their disappearance.

Reduction of elevated P_{CO_2}. Arrhythmias were permitted to continue for from 3 to 25 minutes before they were terminated by reducing the P_{CO_2}. As P_{CO_2} declined the number of ventricular foci, and the frequency of ventricular extrasystoles progressively diminished. In one subject the return to normal rhythm was dramatic (fig. 2). The P_{CO_2} at which ventricular arrhythmias disappeared was 72 mm Hg (S.D. = 16). Sinus rhythm was re-established in 80 per cent of cases when P_{CO_2} had reached 60 mm Hg. An increase in the severity of the arrhythmia during the correction of hypercarbia was not observed. The rate of decrease of P_{CO_2} ranged from 5 to 25 mm Hg/min.

Heart Rate and Arterial Blood Pressure.

These data are summarized in table II.

Normal P_{CO_2}. Before induction of anaesthesia the heart rate in the fifteen subjects averaged 78 beats per minute, and the arterial pressure 91 mm

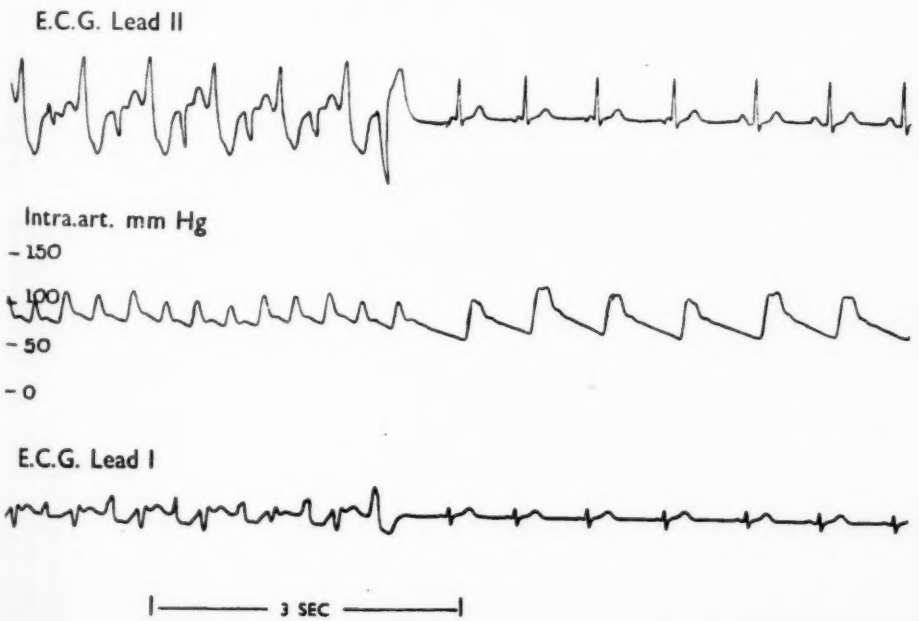

FIG. 2

Reversion of ventricular to sinus rhythm during P_{CO_2} reduction.

TABLE II

Haemodynamic Changes during Halothane Anaesthesia.

Subject			Percent halothane inspired	Mean arterial blood pressure (mm Hg)				Heart rate (beats minute)				
No.	Age	Sex		Before anaesthesia	Stable anaesthesia (Pco_2 normal)	Hypercarbia (sinus rhythm)	Post-hypercarbia (minimum pressure)	Before anaesthesia	Stable anaesthesia (sinus rhythm)	Hypercarbia (sinus rhythm)	Post-hypercarbia	Pco_2 during hypercarbia (mm Hg)
1	30	F	1.9	90 a, b	80	89	80	100	70	90	90	65
2	16	F	2.5	92 a, b	62	56	46	84	63	70	75	92
3	42	F	2.5	90 a, b	72	83	56 b, c	95	79	90	100	135
4	33	F	1.4	70 a, b	60	70	55 b	90	104	114	122	96
5	23	F	1.8	90 a, b	66	66	57	78	65	80	75	95
6	36	F	2.5	90 a, b	104	104	98 b	85	75	81	66	62
7	17	M	2.2	88 a, b	74	70	61 b	70	63	69	78	83
8	32	F	2.0	80 a, b	47 a, b	67 a, b	57 a, b	80	80	96	95	76
9	49	F	1.1	133 a, b	129	100	47 b, c	80	85	66	70	116
10	35	F	2.2	88	45	88	36	86	62	72	74	140
11	22	F	2.2	96	72	77	69	62	60	76	76	73
12	39	F	1.0	77	68	100	85 b	69	66	86	87	72
13	33	F	2.0	70 a, b	64	70	64	75	82	92	94	76
14	15	F	2.2	98	40	30	—	63	68	80	—	—
15	45	F	2.2	120	70	76	73	66	58	62	99	98
Average			2.0	91	70	76	63	79	72	82	86	91

a = auscultatory blood pressure.

b = mean arterial blood pressure calculated as diastolic plus one-third pulse pressure.

c = blood pressure still falling when halothane concentration was decreased.

Hg. Some subjects were apparently apprehensive; tachycardia was the rule in these individuals, although blood pressure was often no greater than when they were visited in the ward on the previous day. The heart rate usually slowed with the induction of anaesthesia, but persistent bradycardia was observed in only nine cases; the others, following transient changes, exhibited either the same or a higher rate than during consciousness. The level of mean arterial blood pressure was reduced by halothane inhalation in all subjects but one, but there was no consistent relationship between the concentration inspired and the degree of hypotension.

Elevated P_{CO_2}. Elevation of the P_{CO_2} during halothane anaesthesia resulted in an increase in heart rate in all subjects but one prior to the onset of cardiac arrhythmias. An increase in arterial pressure was less frequent, occurring in only nine cases. A *reduction* in pressure occurred in four, and in one individual (No. 14) the inhalation of carbon dioxide produced hypotension so profound that its administration was terminated.

When ventricular extrasystoles were present, the heart rate was significantly higher than when they were not, and it averaged 96 beats per minute. A rate of 180 beats per minute was attained in each of the two subjects who developed multifocal ventricular tachycardia. The mean arterial pressure was not consistently altered by the development of these arrhythmias. Although the premature beats usually produced effective ventricular contractions, they were sometimes nearly ineffective in producing a pulse (fig. 3).

There was a significant relationship ($p < 0.01$) between the mean arterial blood pressure just before carbon dioxide inhalation and the P_{CO_2} "arrhythmia threshold" in that the threshold was higher, the lower the blood pressure (fig. 4). In two subjects in whom blood pressure was low during stable anaesthesia, ventricular arrhythmias could not be produced by carbon dioxide inhalation.

One subject had a mean blood pressure of 45 mm Hg during the stable state of anaesthesia. No ventricular arrhythmias occurred despite inhalation of a concentration of carbon dioxide over 140 mm Hg. The concentration of halothane was then reduced and following a second period of stable anaesthesia when the mean arterial blood

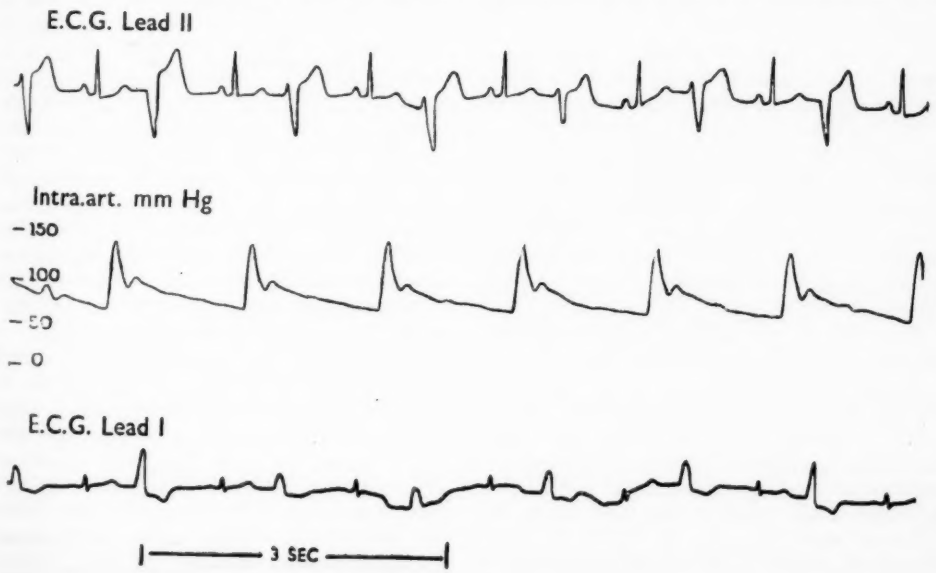

FIG. 3
Ineffective pressure pulses produced by ventricular extrasystoles. Compare normal beats.

TABLE II
Haemodynamic Changes during Halothane Anaesthesia.

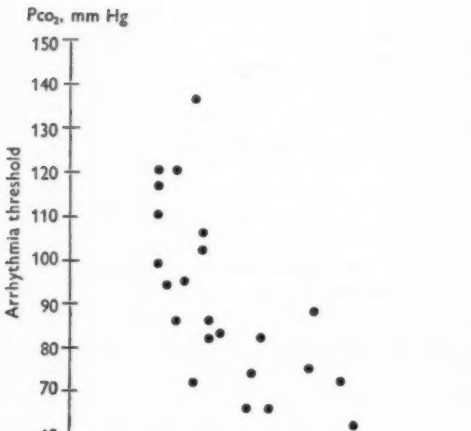

FIG. 4

Arrhythmia threshold (P_{CO_2}) in relation to mean arterial blood pressure (M.A.B.P.).

pressure was higher (60 mm Hg), elevation of the P_{CO_2} to 110 mm Hg gave rise to ventricular extrasystoles.

In the other subject the mean arterial blood pressure was 40 mm Hg during stable anaesthesia and the addition of carbon dioxide reduced this to 30 mm Hg at a point when the P_{CO_2} was 82 mm Hg. In view of the hypotension, the concentration of halothane was reduced. In the period of stable anaesthesia which followed this the mean arterial blood pressure was 60 mm Hg and ventricular premature contractions were subsequently produced by elevating P_{CO_2} to 117 mm Hg.

Reduction of elevated P_{CO_2}. Following withdrawal of carbon dioxide and the return to normocarbia the minimum arterial blood pressure averaged 69 mm Hg. Minima occurred between 2 and 10 minutes after the return of P_{CO_2} to normal. A marked decrease in pressure, compared to observations made just before the induction of hypercarbia, was observed in one patient (No. 9), and a moderate reduction occurred in two other cases (Nos. 2 and 3). With these exceptions no patient exhibited a reduction in pressure as great as 10 mm Hg compared with that measured in the period of stable anaesthesia before carbon dioxide inhalation. In two cases the pressure was significantly higher after carbon dioxide inhalation than it had been before. When the minimum blood pressure was observed, the average heart rate was 86 beats per minute, and individual values were almost uniformly higher than before hypercarbia.

DISCUSSION

In this study normal sinus rhythm (or minor variations of it) was always present during halothane inhalation, provided that normal alveolar ventilation was maintained. Bradycardia was common, but the only evidence of pacemaker displacement was a slight change in the shape of the P wave in some cases, indicating a shifting site of impulse formation within the sino-atrial node. Atrioventricular nodal rhythm, ventricular extrasystoles, and other abnormalities of rhythm occurred only when respiratory obstruction or depression was present, or when alveolar carbon dioxide concentration was deliberately elevated by the administration of this gas.

These results contrast with observations made during cyclopropane anaesthesia (Lurie et al., 1958), when ventricular arrhythmias could be produced at normal levels of P_{CO_2} merely by increasing the concentration of cyclopropane respired. Possible explanations for this difference between the two agents will be suggested below.

Burns and co-workers (1957) reported that ventricular premature contractions occurred when the concentration of halothane was high and that a reduction of vapour strength abolished them. However, these authors found that halothane reduced the minute and, even more, the tidal volume of respiration. This effect was more marked at high concentrations of halothane. It seems inevitable that the alveolar P_{CO_2} increased when the vapour strength of halothane was elevated, and it is likely that this could have caused cardiac arrhythmias which were incorrectly attributed to halothane per se. Unfortunately, the P_{CO_2} was not estimated. Much the same thing can be said for the results of Johnstone (1956) and Hudon et al. (1957). This is not to say that hypercarbia is the only cause of cardiac arrhythmia during halothane inhalation; but it is a likely cause until its existence can be eliminated.

In the present study, elevation of the end-expired P_{CO_2} initiated cardiac arrhythmias in every subject. This result is in agreement with those of Johnstone (1956) and Stephen et al. (1957) who, however, did not measure the degree of hypercarbia necessary to initiate abnormalities of rhythm. In our subjects the end-expired P_{CO_2} at which a ventricular arrhythmia appeared ranged from 62 to over 140 mm Hg (average 92 mm Hg). This "threshold" was not influenced by the depth of halothane anaesthesia in any consistent way. The arrhythmias persisted while carbon dioxide concentration remained at the "threshold" level or above, further elevation did not increase their severity, and reduction of the P_{CO_2} led to their abolition.

While these findings resemble those previously reported during cyclopropane anaesthesia (Lurie et al., 1958), there are important differences. First, the P_{CO_2} threshold for the production of arrhythmias during cyclopropane anaesthesia (mean 74, range 44–107 mm Hg) was significantly lower than during halothane anaesthesia. Second, the higher the concentration of cyclopropane inspired, the lower was the alveolar P_{CO_2} at which arrhythmias occurred. Third, arrhythmias, instead of disappearing when P_{CO_2} was reduced, sometimes increased in frequency. An exacerbation of arrhythmia typically occurred only when the P_{CO_2} was reduced at a rapid rate (i.e. over 40 mm Hg/min). Since in the present study the maximum rate of fall was only 25 mm Hg/min this difference may be more apparent than real. Even so, exacerbation occurred occasionally during cyclopropane anaesthesia at rates of reduction as low as 15 mm Hg/min.

An explanation for these differences between halothane and cyclopropane has been sought by us in studies of the sympatho-adrenal responses to anaesthesia and hypercarbia (Price et al., 1959). Many anaesthetics, including halothane and cyclopropane, "sensitize" the myocardium to the actions of the catechol amines. Since one of these actions is the production of arrhythmias, it is evident that disorders of rhythm can result, if the anaesthetic itself or some change associated with its inhalation (e.g. hypercarbia or hypoxia) calls forth sympatho-adrenal activity. This idea is not new, for it resembles Levy's (1911) views on chloroform. What is new is the existence of methods adequate to estimate the degree of sympathetic response from microchemical analysis of peripheral blood (Price and Price, 1957). Cyclopropane inhalation was found by us to increase the concentration of noradrenaline in plasma when the P_{CO_2} was normal. Halothane did not do so (Price et al., 1959). Carbon dioxide inhalation during cyclopropane anaesthesia produced a further increase in noradrenaline concentration, and adrenaline also appeared in the plasma in large amounts (Price et al., 1958). During halothane anaesthesia the sympatho-adrenal response to hypercarbia was weak. Reduced reactivity of the sympathetic nervous system during halothane anaesthesia thus may explain the absence of ventricular arrhythmias at normal P_{CO_2} levels, as well as the relatively high P_{CO_2} required for their production. The synergism between cyclopropane and carbon dioxide in producing arrhythmias, in contrast, could result from the fact that both substances produce a common effect, namely increased sympathetic nervous discharge.

The high P_{CO_2} needed to produce arrhythmias during halothane administration may be related also to the arterial hypotension which this anaesthetic commonly produces, for it has been shown that ventricular arrhythmias are more easily produced when blood pressure is high than when it is low (Moe et al., 1948). In addition to acting centrally to produce stimulation of the sympathetic nervous system, carbon dioxide acts peripherally causing vasodilatation and decreased myocardial contractility. When the sympathetic response is reduced, as it may be by halothane, the peripheral effect may predominate, resulting in arterial hypotension. In four of our subjects the blood pressure response to hypercarbia was indeed that of reduction of pressure, and marked increases in pressure rarely occurred. These facts may also help in explaining the high arrhythmia threshold.

The small degree of post-hypercarbic hypotension usually found during halothane anaesthesia is similar to the experience of Lurie and associates (1958) during cyclopropane administration, and also to that of Sechzer et al. (1959), in conscious man. Observations made in this laboratory during the past several years make us believe that when hypotension occurs in surgical patients following the correction of respiratory

acidosis, the degree of blood pressure reduction is related as much to such factors as reduction of anaesthetic concentration, blood loss, and motion, as to P_{CO_2} reduction.

SUMMARY

The effects of halothane inhalation on arterial blood pressure and cardiac rate and rhythm during normocarbia and hypercarbia were studied in fifteen subjects. The electrocardiogram, end-expired carbon dioxide tension, and brachial arterial pressure were recorded continuously. Hypercarbia was produced by carbon dioxide inhalation. Significant abnormalities of cardiac rhythm did not occur except during airway obstruction or carbon dioxide inhalation. Arterial pressure was consistently, and heart rate inconsistently, reduced. When carbon dioxide was administered, abnormal cardiac rhythms were produced in every case. The average P_{CO_2} at which these arrhythmias occurred was 92 mm Hg. Hypertension during carbon dioxide inhalation was not marked; it was sometimes absent, and hypotension occasionally occurred. Return of the elevated P_{CO_2} to normal was associated with hypotension which, however, was pronounced in only three cases.

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REBREATHING DURING CONTROLLED RESPIRATION WITH THE MAGILL ATTACHMENT

BY

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THE Magill attachment (Mapleson, 1954, type "A") was originally designed to provide a reservoir of anaesthetic gas mixture which could be respired by the patient without dilution by room air and without appreciable rebreathing. Molyneux and Pask (1951) studied the flow of gas in such a system and demonstrated that there was a correlation between tidal volume and the fresh gas inflow which was necessary to prevent rebreathing. Mapleson (1954), in a theoretical analysis, concluded that rebreathing of alveolar gas could be prevented by ensuring that the fresh gas inflow was at least equal to the patient's minute volume. Woolmer and Lind (1954) and Bracken and Sanderson (1955), using experimental models, came to much the same conclusion. Davies, Verner and Bracken (1956) confirmed these results in patients undergoing surgery.

Many anaesthetists today, however, perform assisted or controlled respiration with the Magill attachment by tightening the expiratory valve and applying intermittent positive pressure to the reservoir bag. Under these conditions the valve opens during the inspiratory phase instead of during late expiration (fig. 1). Such a change might be expected to alter the efficiency of carbon dioxide elimination from the circuit. To determine whether this was so, a model lung was designed which enabled the Magill attachment to be studied under conditions of "spontaneous" respiration and "intermittent positive pressure respiration" (I.P.P.R.) (fig. 3).

The use of a model in the first instance was considered justified as it eliminated most of the variables encountered during human experiments.

PART I

Experimental Studies (fig. 3)

The reservoir bag of the Magill attachment was enclosed in an aspirator bottle, the anaesthetic

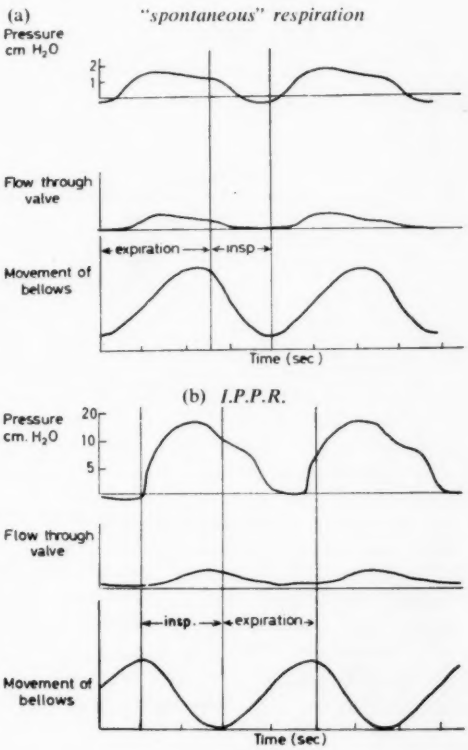

FIG. 1

Recordings made with the apparatus shown in figure 2 to show the relationship between the phase of respiration and the flow of gas through the expiratory valve.

(*a*) "Spontaneous" respiration. (*b*) "I.P.P.R."

Upper tracing: Pressure within the expiratory valve mount.

Middle tracing: Rate of gas flow through valve.

Lower tracing: Movement of Beaver respirator bellows.

Time trace in seconds.

FIG. 2

Diagram of apparatus used to obtain traces shown in figure 1. The gas expelled from the expiratory valve passes to the atmosphere through a constricted tube. The pressure in the chamber surrounding the valve gives an indication of flow rate and is recorded on the drum.

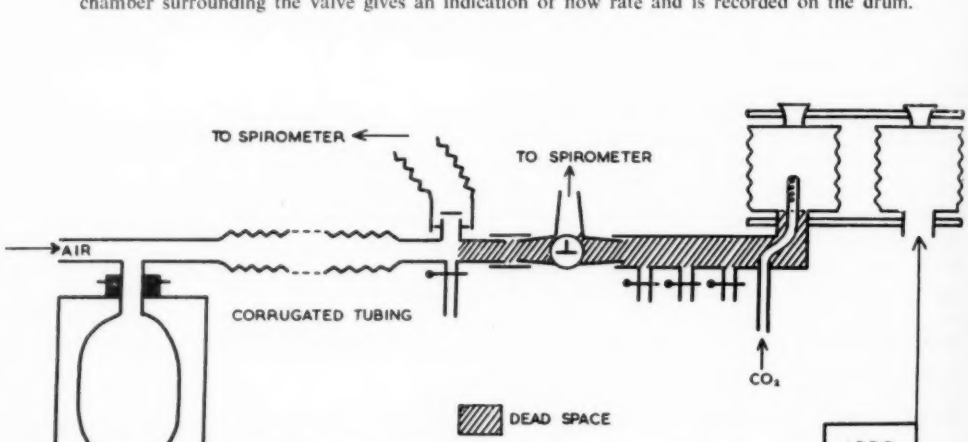

FIG. 3

Diagram of the apparatus used to study rebreathing with the Magill attachment.

machine being replaced by a cylinder of compressed air with reducing valve and calibrated flowmeter. The patient's lungs were represented by a rubber bellows into which was introduced a continuous flow of 200 ml of carbon dioxide per minute. The expiratory valve mount was connected to the bellows by a rubber tube representing the patient's deadspace. Several side tubes were tapped into the deadspace for sampling purposes. An exactly similar bellows was mounted parallel to the "lung" in the free edge of a hinged frame. By connecting the output from a Beaver Mk. II respirator to this bellows a parallel change in volume of the "lung" bellows was induced, thus simulating "spontaneous" respiration. By connecting the respirator to the bottle, intermittent positive pressure could be applied to the reservoir bag.

The tidal volume was measured in the earlier experiments by manipulating a three-way tap, situated between the deadspace tube and expiratory valve mount, so as to deflect the expired gas into a spirometer. With a little practice readings could be obtained which varied by less than 30 ml. The mean of three readings was taken. The volume of the deadspace (tubing, tap and connections) was measured by water displacement and adjusted to 150 ml. The bellows were made of stiff rubber and kept partially expanded by a fixed stop. In this position the lung bellows had a "functional residual capacity" of 900 ml. Over the range of inflation 0–1,300 ml the static compliance was found to be linear and equal to 65 ml/cm H_2O pressure. Thus inflation pressures required were similar to those used in anaesthesia.

The apparatus was tested for leaks before and after each run by collecting the gases from the expiratory valve and comparing the volume expired in 1 minute during "spontaneous" respiration and during maximum intermittent positive pressure respiration. If there was a difference of more than 200 ml/min (2 per cent) the run was excluded from the results.

The mass spectrometer was used for continuous analysis of gas samples taken from one of the side tubes in the "deadspace". The volume of gas withdrawn from the system was less than 30 ml/min and the analyses were accurate to within ±1 mm Hg partial pressure of carbon dioxide. "Alveolar" plateaux, similar to those found in patients were regularly obtained, thus demonstrating that mixing in the bellows was satisfactory.

Results

"Alveolar" carbon dioxide levels during "spontaneous" respiration and "I.P.P.R."

The air inflow was kept constant at 10 l./min, the carbon dioxide input at 200 ml/min and the respiratory rate at 15/min. The deadspace was 150 ml. Samples were taken from the middle tube in the "deadspace". The "alveolar" carbon dioxide levels recorded at varying tidal volumes are shown in table I. Figure 4 shows the results expressed graphically.

A theoretical "alveolar" carbon dioxide percentage, assuming no rebreathing of the expired gas which passed beyond the confines of the "deadspace" tubing (vol. 150 ml) was calculated from the formula:

$$\text{Alveolar carbon dioxide } \% = \frac{\text{carbon dioxide input (ml)} \times 100}{(\text{tidal volume (ml)} - \text{deadspace (ml)}) \times \text{respiratory rate}}$$

During "spontaneous" respiration at normal tidal volumes the observed "alveolar" carbon dioxide levels approximated to the theoretical values.* At low tidal volumes there was poor agreement. This was due, as in the human subject, to the "cone front" effect of the advancing gases (Briscoe et al., 1954).

During "I.P.P.R." at tidal volumes below 300 ml the "alveolar" carbon dioxide levels were close to the theoretical curve plotted for "spontaneous" respiration. At greater tidal volumes with "I.P.P.R." the concentrations of carbon dioxide in the "alveolar" air were higher than during "spontaneous" respiration; this difference was approximately 3 per cent at the highest tidal volumes recorded. The importance of such a change can be better appreciated if it is realized that an increase of 3 per cent in the alveolar carbon dioxide concentration in the model, at a tidal volume of 750 ml, is equivalent to an apparatus deadspace of nearly 400 ml.

The majority of anaesthetists, when performing manual inflation of the lungs, produce pressure curves similar to those produced by the respirator used in this experiment. Some, however, produce

*See appendix for sources of error.

TABLE I

Performance of Magill attachment during "Spontaneous" respiration

Gas inflow 10 l./min. Carbon dioxide input 200 ml/min. Deadspace 150 ml. Respiratory rate 15 /min. Theoretical "alveolar" carbon dioxide calculated on the basis of no rebreathing.

Tidal volume (ml)	"Alveolar" CO_2 (%)	Theoretical alveolar CO_2 (%)	Difference
780	1.33	2.12	−0.79
767	2.76	2.16	+0.60
760	2.76	2.19	+0.57
703	3.16	2.40	+0.76
643	3.48	2.70	+0.78
620	3.03	2.83	+0.20
620	2.76	2.83	−0.07
610	3.82	2.90	+0.92
600	3.95	2.96	+0.99
593	3.42	3.01	+0.41
590	3.16	3.03	+0.13
580	3.68	3.10	+0.58
530	4.48	3.51	+0.97
527	3.55	3.53	+0.02
527	4.48	3.55	+0.95
520	3.29	3.60	−0.31
510	3.82	3.70	+0.12
500	4.48	3.81	+0.67
490	3.68	3.92	−0.24
466	5.53	4.21	+1.32
443	5.40	4.54	+0.86
427	4.48	4.80	−0.32
370	5.26	6.05	−0.79
343	7.63	6.92	+0.71
336	8.42	7.15	+1.27
305	7.63	8.60	−0.97
300	9.21	8.88	+0.33
283	10.78	10.02	+0.76
180	11.30	44.30	−33.00
176	14.00	51.20	−37.20

Performance of Magill attachment during "I.P.P.R."

Gas inflow 10 l./min. Carbon dioxide input 200 l/min. Deadspace 150 ml. Respiratory rate 15 /min. Theoretical carbon dioxide percentage calculated on the basis of no rebreathing.

810	4.74	2.02	2.72
760	4.74	2.18	2.56
733	5.92	2.28	3.64
720	5.40	2.33	3.07
646	5.26	2.69	2.57
600	5.79	2.96	2.83
590	6.05	3.03	3.02
579	6.58	3.11	3.47
567	6.06	3.19	2.87
550	5.40	3.33	1.07
523	6.58	3.57	3.01
510	5.50	3.70	1.80
470	7.87	4.16	3.71
450	6.98	4.44	2.54
410	7.49	5.12	2.37
405	7.11	5.23	1.88
350	9.47	6.66	2.81
310	9.21	8.35	0.86
280	11.82	10.25	0.57
265	11.55	11.60	−0.05
260	13.16	12.10	1.06
250	12.62	13.33	−0.71

an inspiratory pressure curve with an almost vertical rise. This was simulated by using a Radcliffe respirator (Russell and Schuster, 1953) in a second series of experiments. Similar results were obtained, suggesting that the shape of the applied pressure curve does not materially effect the results.

Effect of altering the inflow of fresh gas.

Table II shows the results obtained.

Decreasing the inflow of fresh gas during "spontaneous" respiration from 10 l./min to 5 l./min produced a rise in the "alveolar" carbon dioxide level. This rise was most marked at the higher tidal volumes recorded. A similar drop in the flow rate of fresh gas during "I.P.P.R." produced a very marked rise in the carbon dioxide level. The reverse effect was obtained by increasing the flow of fresh gas; but even with a 20 l./min inflow the "alveolar" carbon dioxide level was still raised above the theoretical value for "spontaneous" respiration. It is therefore concluded that although a fresh gas inflow equal to the patient's minute volume will reduce rebreathing to a reasonable level with "spontaneous" respiration, the fresh gas inflow required during "I.P.P.R." to produce the same effect is so large as to be impracticable.

TABLE II

Effect of altering the inflow of fresh gas at varying tidal volumes

Carbon dioxide input 200 ml/min. Respiratory rate 15 /min. Deadspace 150 ml.

Tidal volume (ml)	Alveolar CO_2 (%) 10 l./min inflow	5 l./min inflow	Difference	Theoretical alveolar CO_2 (%)
Spontaneous respiration				
590	3.16	4.22	1.06	3.03
520	3.29	4.61	1.32	3.60
490	3.68	4.48	0.80	3.92
370	5.26	5.66	0.40	6.05
290	6.98	7.63	0.65	9.53
180	11.30	11.82	0.52	44.30
I.P.P.R.				
646	5.26	9.08	3.82	2.69

I.P.P.R. Effect of increasing the gas inflow

Gas inflow (l./min)	Tidal volume (ml)	Alveolar CO_2 (%)	Theoretical alveolar CO_2 (%)
10	567	6.06	3.19
15	567	4.97	3.19
20	567	3.93	3.19

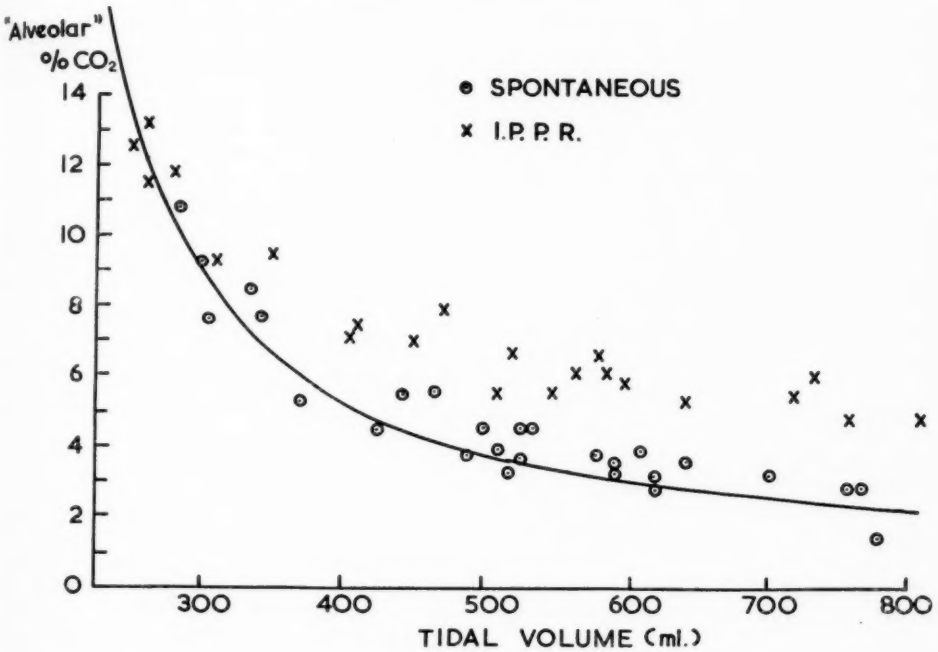

FIG. 4

Graph of results. Theoretical alveolar carbon dioxide concentration (no rebreathing) shown by continuous line. Gas inflow 10 l./min, CO_2 input 200 ml/min. Respiratory rate 15/min. Ordinate: $\%CO_2$ in "alveolar" sample. Abscissa: Tidal volume in ml

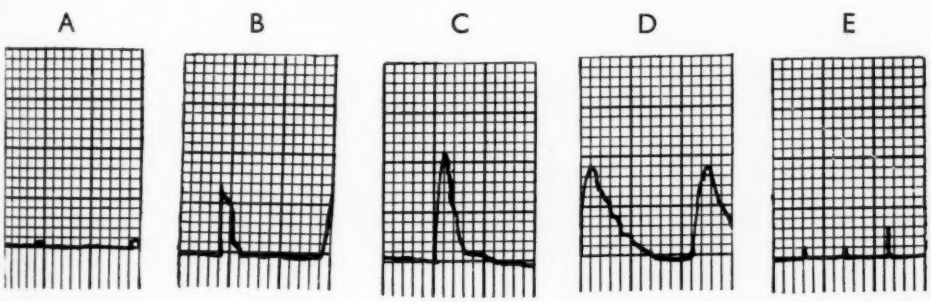

FIG. 5

Gas samples taken from varying positions within the corrugated tubing of the Magill attachment during "spontaneous" respiration.

Ordinate: $\%CO_2$ as recorded by the mass spectrometer.
Abscissa: Time (A, B, C, D): Each vertical line = 0.4 sec. (E): each vertical line = 1.0 sec.

(A) T.V. 590 ml. Sample 50 cm (20 inches) proximal to valve. End-expiratory CO_2 = 0.13%.
(B) T.V. 590 ml. Sample 30 cm (12 inches) proximal to valve. End-expiratory CO_2 = 1.58%.
(C) T.V. 590 ml. Sample 10 cm (4 inches) proximal to valve. End-expiratory CO_2 = 2.89%.
(D) T.V. 763 ml. Sample 30 cm (12 inches) proximal to valve. End-expiratory CO_2 = 2.37%.
(E) T.V. 347 ml. Tip of sampling catheter gradually approximated to valve. A carbon dioxide peak was first detected at a distance of 10 cm (4 inches) from the valve.

The inspired carbon dioxide concentration.

Since the carbon dioxide input was kept constant any increase over the theoretical "alveolar" carbon dioxide level must have been due to the presence of carbon dioxide in the inspired gas. A third series of experiments was therefore performed with a pneumotachograph substituted for the three-way tap and the sampling point situated 5 cm (2 inches) from the centre of the valve, between it and the pneumotachograph. The readings from the pneumotachograph were electrically integrated so that inspiratory volume could be recorded simultaneously with the carbon dioxide concentrations. Continuous records were made at different tidal volumes during "I.P.P.R." and "spontaneous" respiration. The record of a single breath was then analyzed at 0.1-second intervals and the inspired carbon dioxide concentration was plotted against volume inspired at the same point of time. A curve was thus produced and by calculating the area under the curve it was possible to determine the total volume of carbon dioxide in each breath of inspired gas. The mean of five breaths was taken at each tidal volume.

During "spontaneous" respiration with a gas inflow of 10 l./min the volume of carbon dioxide in the inspired gas was small (table III). The volume was increased if the tidal volume was increased or if the fresh gas inflow was reduced. The much larger volumes of carbon dioxide contained in the inspired gas during "I.P.P.R." are also shown in this table. It will be noted that there was a good agreement between the expected "alveolar" carbon dioxide concentrations (calculated from the volume of carbon dioxide in the inspired gas) and the observed value.

Samples from corrugated tube.

The presence of carbon dioxide in the corrugated tube at the end of expiration was confirmed by sampling at various points along the tube (table IV and fig. 5). It then became apparent that the length of tube containing carbon dioxide at the end of expiration bore a rough relation to the tidal volume. The peak carbon dioxide level at the end of expiration was observed to become progressively less as the distance from the valve increased—presumably due to mixing with fresh gas left in the corrugated tube.

Samples of gas eliminated from the system.

The results detailed so far suggest that during "I.P.P.R." the expired gas passes en bloc into the corrugated tube (fig. 6). On compressing the bag a proportion of this gas is forced out through the valve and the rest is driven into the bellows by the flow of fresh gas. If there are no leaks and the lung deflates completely the total volume of gas forced out through the valve must be equal to the total gas flowing into the system.

To substantiate the theory illustrated in figure 6 an attempt was made to determine the concentration of carbon dioxide in the gas flowing through the valve and to compare this with the carbon dioxide concentration in the gas passing

TABLE III

The relationship between volume of carbon dioxide in the inspired gas and the observed "alveolar" Carbon Dioxide Concentration

CO_2 input 200 ml/min. Respiratory rate 15/min. Deadspace 150 ml.

Inflow of fresh gas (l./min)	Tidal volume (ml)	Volume of CO_2 in inspired gas (ml/min)	Expected alveolar CO_2 concentration calculated from volume of CO_2 in inspired gas (%)	Observed alveolar CO_2 concentration (%)	Theoretical alveolar CO_2 concentration (assuming no rebreathing) (%)
Spontaneous respiration					
10	300	2.2	9.00	9.21	8.88
5	300	15.3	9.56	9.80	8.88
10	590	4.9	3.11	3.16	3.03
5	590	80.1	4.24	4.22	3.03
I.P.P.R.					
10	720	256	5.35	5.40	2.33
10	560	205	6.61	6.50	3.25
10	320	71	10.60	10.36	7.84

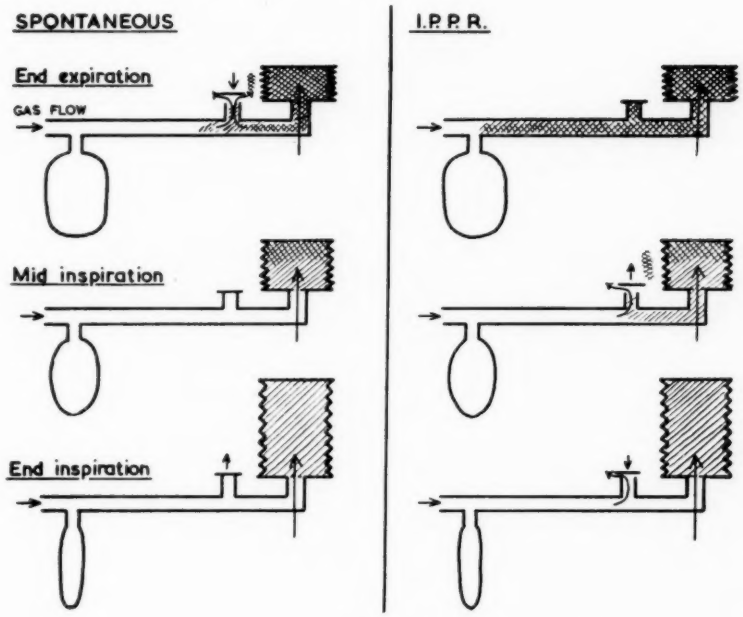

FIG. 6

Diagram to show the distribution of carbon dioxide-rich gas (shaded) during the various phases of respiration.

TABLE IV

Concentrations of carbon dioxide in corrugated tubing of Magill attachment at end-expiration

Gas inflow 10 l./min. CO_2 input 200 ml/min. 1 inch = 2.5 cm.

I.P.P.R.

Tidal volume (ml)	Minute volume (l.)	Sample point	End-expiratory CO_2 (%)
567	8.5	Deadspace	6.06
567	8.5	6 inches proximal to expiratory valve	5.95
567	8.5	14 " "	5.24
567	8.5	20 " "	4.26

Spontaneous respiration

Tidal volume (ml)	Sample point	End-expiratory CO_2 (%)
590 (fig. 5 "A")	20 inches proximal to expiratory valve	0.13
590 (fig. 5 "B")	12 " "	1.58
590 (fig. 5 "C")	4 " "	2.89
763 (fig. 5 "D")	12 " "	2.37
347	12 " "	0

under the valve (fig. 7). In this experiment, the extremely fine tip of the sampling catheter was placed so that it rested on the edge of one of the expiratory valve ports, exactly opposite the valve seating and about 2 mm away from the edge of the valve.

Considering spontaneous respiration first (C, D, E, F, fig. 7), it is evident that at a high tidal volume the concentration of carbon dioxide in the gas passing out through the valve (D) is the same as that in the alveolar plateau (C). There is a sudden peak as the valve opens and this is then lowered by an increasing proportion of fresh gas. At a lower tidal volume (E, F) the peak is only two-thirds of the alveolar concentration and the issuing gas (alveolar and fresh gas) is diluted quickly by more fresh gas. This agrees with the finding that expired gas containing carbon dioxide passes further into the corrugated tube at the end of a large expiration than at the end of a smaller one.

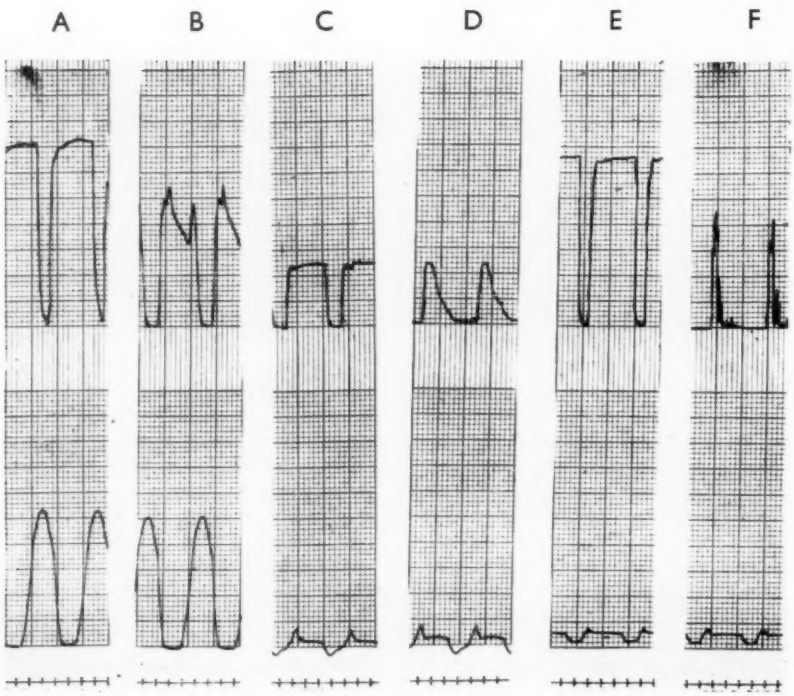

FIG. 7

Upper tracings: Mass spectrometer records of CO_2 content of gas flowing out through the expiratory valve and of deadspace gas samples taken at the same tidal volumes. Middle tracing: Pressure under the expiratory valve. Lower tracing: Time in seconds.

	Respiration	Tidal volume	Sampling point
(A)	I.P.P.R.	355 ml	Deadspace
(B)	I.P.P.R.	355 ml	Valve
(C)	Spontaneous	710 ml	Deadspace
(D)	Spontaneous	710 ml	Valve
(E)	Spontaneous	355 ml	Deadspace
(F)	Spontaneous	355 ml	Valve

The pattern is more complicated during "I.P.P.R.". The "dog ears" recorded in (B), when interpreted in conjunction with the pressure trace, can probably be explained as follows. The smaller peak corresponds to the opening of the valve and the elimination of alveolar gas mixed with some fresh gas. This is followed by fresh gas from the upper part of the corrugated tube and the reservoir bag. The subsequent (and taller) peak occurs during expiration as alveolar gas passes under the valve, which does not close until the pressure under it has fallen considerably. This peak then undergoes a gradual fall as the carbon dioxide mixes with the room air outside the valve. A similar curve was obtained at larger tidal volumes, when the peak concentration more closely approached the alveolar concentration.

PART II

Clinical Studies

Confirmatory studies were carried out on three volunteers who required a general anaesthetic for minor surgery on the head and neck. Premedication consisted of pethidine 75 mg. and atropine 0.6 mg subcutaneously 1 hour before operation. Anaesthesia was induced with a minimal dose of thiopentone followed by suxamethonium. The lungs were inflated with oxygen and the larynx

and trachea were thoroughly sprayed with 4 per cent lignocaine. A large-bore cuffed endotracheal tube was inserted and an air tight fit was obtained by inflating the cuff. The lungs were gently inflated with oxygen until spontaneous respiration returned. An intravenous dose of pethidine (30–50 mg) was then given slowly and further small doses of thiopentone (25 mg) were added at intervals to maintain unconsciousness. A total dose of 600 mg of thiopentone was never exceeded. Surgery was not commenced until the studies had been completed.

When spontaneous respiration recommenced the patient was connected to the Magill attachment, the arrangement being similar to that shown in figure 3. Air from a cylinder was led into the apparatus at a rate of 10 l./min. A pneumotachograph was interposed between the expiratory valve mount of the Magill attachment and the patient. It was connected to a Cobb suction connection in the endotracheal tube by a short length of rubber tubing which was pierced at one point by the sampling catheter from the mass spectrometer. The total deadspace of patient and apparatus was estimated to be under 200 ml.

The patient was first allowed to breathe spontaneously until the recordings of tidal volume, respiratory rate and alveolar P_{O_2} and P_{CO_2} had demonstrated the existence of a steady state for at least 2 minutes. During this time the cycling of the Beaver respirator was adjusted to equal the respiratory rate of the patient. Apnoea was then induced with suxamethonium and the respirator was connected to the bottle. The tidal volume was quickly adjusted to equal that measured during the steady period of spontaneous respiration by manipulating the volume control on the respirator and the expiratory valve on the Magill attachment. Care was taken to prevent over-distension of the bag during expiration. Apnoea was then maintained with further doses of suxamethonium until tidal volume and alveolar P_{O_2} and P_{CO_2} levels had been steady for at least 2 minutes. Finally the tidal volume was greatly increased and further observations made on the composition of the alveolar gas.

Results (table V)

In all three patients there was a fall in alveolar P_{O_2} and a rise in alveolar P_{CO_2} when spontaneous respiration was replaced by I.P.P.R. at the same or slightly greater tidal volume. In the first case (B.R.F., fig. 8) the fall in alveolar P_{O_2} was so marked that cyanosis occurred. This was abolished by increasing the tidal volume from 510 ml to 740 ml, but even at this tidal volume the alveolar

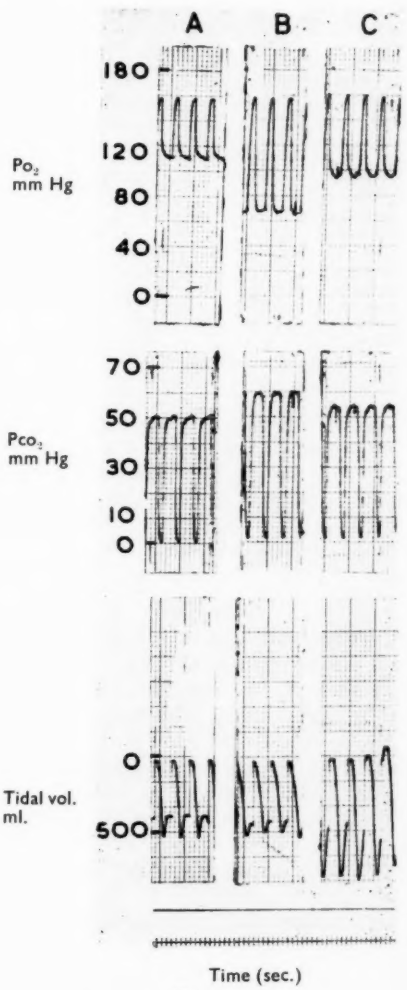

FIG. 8

Mass spectrometer tracings from Case B.R.F.
(A) Spontaneous respiration.
(B) I.P.P.R. at same tidal volume.
(C) I.P.P.R. at increased tidal volume.

TABLE V

Showing alveolar P_{O_2} and P_{CO_2} during spontaneous respiration and I.P.P.R. with the Magill attachment (see figure 8).

PATIENT B.R.F. Respiration Rate 16/min	Tidal Vol. ml	Alveolar P_{O_2} mm Hg	Alveolar P_{CO_2} mm Hg
Spontaneous	490	110	50
I.P.P.R.	510	65 still falling	60 still rising
I.P.P.R.	740	95	56
PATIENT J.W. Respiration Rate 12/min	**Tidal Vol. ml**	**Alveolar P_{O_2} mm Hg**	**Alveolar P_{CO_2} mm Hg**
Spontaneous	580	90	49
I.P.P.R.	620	78	53
I.P.P.R.	810	94	51
PATIENT A.B. Respiration Rate 14/min	**Tidal Vol. ml**	**Alveolar P_{O_2} mm Hg**	**Alveolar P_{CO_2} mm Hg**
Spontaneous	380	95	50
I.P.P.R.	410	76	56
I.P.P.R.	800	96	51

P_{CO_2} did not fall below 56 mm Hg. In the other two cases the changes were less marked but showed the same general trend. In none of the cases was it possible to lower the alveolar P_{CO_2} during I.P.P.R. below the level which had existed during spontaneous respiration.

CONCLUSION

These studies show that when the Magill attachment is used the volume of carbon dioxide which escapes through the expiratory valve is determined by several factors. The total volume of gas that passes through the valve at each breath is dependent on the respiratory rate and the total inflow into the system. The concentration of carbon dioxide in this gas depends on the concentration of carbon dioxide in the gas lying under the valve during the period when the valve is open.

With "spontaneous" respiration, providing the gas inflow is in excess of the minute volume, the amount of carbon dioxide eliminated is dependent on the "alveolar" carbon dioxide level. This in turn bears a direct relation to the alveolar ventilation and the volume of carbon dioxide entering the "lungs".

With "I.P.P.R." two additional factors are present. The first is the extent of rebreathing, which is determined by the tidal volume and the fresh gas inflow. The second factor is the time during which the valve remains open. This depends on the pressure necessary to produce a given tidal volume.

In view of the complexity of these factors and the tremendous increase in apparatus dead space which results when the Magill attachment is used for "I.P.P.R." at normal or large tidal volumes, it is recommended that the apparatus should never be used during assisted or controlled ventilation. The patient should be switched to an efficient absorption system whenever artificial assistance to respiration becomes necessary. The interposition of a nonrebreathing valve between expiratory valve and patient provides a cheaper and even more efficient alternative which deserves to be much more widely used.

SUMMARY

The behaviour of the Magill attachment during "spontaneous" respiration and "I.P.P.R." has been tested on an experimental model and in patients.

It has been shown that during "I.P.P.R." with this system rebreathing can only be prevented by using a very large inflow of fresh gas.

The degree of rebreathing when expressed as apparatus deadspace may reach a figure of 400 ml at tidal volumes common to adult male patients.

A plea is made for the more widespread use of nonbreathing valves.

APPENDIX

The scatter noted in the results may be attributed to three sources:

(1) Variation in the alveolar level of carbon dioxide. The alveolar percentage varied by 0.1–0.3 per cent from breath to breath even when a steady state had been achieved. This was probably due to irregularity in the action of the valve.

(2) Errors in measuring tidal volume—up to 30 ml.

(3) Errors due to variation in carbon dioxide input. The only carbon dioxide flowmeter available was the standard 2-litre flowmeter fitted to the Gillies anaesthetic machine. Leaks were eliminated by taking a direct lead from the top of the flowmeter to the lung bellows. At the low flows used, however, the rotameter bobbin was inclined to wobble, and calibrations on a spirometer with the bobbin set at 200 ml/min. varied between 185 and 220 ml. A further error tended to occur during "I.P.P.R." since the bobbin was depressed during the positive pressure phase. Thus the

carbon dioxide input was probably lower than during "spontaneous" respiration. Correction of this error would tend to magnify the difference recorded between "I.P.P.R." and "spontaneous" respiration.

ACKNOWLEDGMENTS

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PLASMA ADRENALINE AND NORADRENALINE LEVELS DURING HAEMORRHAGE INDUCED AFTER CHLORPROMAZINE INJECTION

BY

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IT has been reported that chlorpromazine protects against irreversible haemorrhagic shock (Overton and DeBakey, 1956; Spurr et al., 1956) when administered before but not after the induction by haemorrhage of hypotension (Gowdey et al., 1957). It has been claimed that chlorpromazine exerts a beneficial effect in these and other conditions of "sympathetic irritation" because of a central sympathetic inhibitory action (for a review of the literature see Hopkin, 1955; Hopkin and Brown, 1958). This implies that chlorpromazine has an "automatic stabilizing" effect of value in the anaesthetized or shocked patient. However, it will be recalled that Holzbauer and Vogt (1954) found that chlorpromazine did not inhibit the stimulation of sympathetic centres (i.e. did not prevent depletion of hypothalamic noradrenaline) induced in cats by morphine injection or by the stress of surgery.

We have previously reported on the plasma and urinary adrenaline and noradrenaline levels during progressive haemorrhage in dogs (Millar and Benfey, 1958). It appeared to be of interest to measure plasma catechol amine levels during haemorrhage induced following chlorpromazine administration.

METHOD

Five dogs were used. Each dog was lightly anaesthetized with thiopentone, the groin infiltrated with a few millilitres of 1 per cent lignocaine solution, and the femoral artery cannulated to allow withdrawal of blood samples and direct measurement of arterial blood pressure by means of a Statham strain gauge and Sanborn recorder. After withdrawal of a blood sample (Sample 1, 40 ml) 5–10 mg/kg of chlorpromazine was injected slowly intravenously. One hour later, a second blood sample (Sample 2, 40 ml) was taken to determine any effect of chlorpromazine on circulating adrenaline and noradrenaline levels. Every 15 minutes thereafter, 10 ml/kg blood samples were withdrawn until circulatory collapse occurred.

Plasma was immediately separated from blood samples by centrifuging, and assays of plasma adrenaline and noradrenaline completed within 24 hours. The chemical method of estimation as originally described (Millar and Benfey, 1958) has been modified for these and other studies and is included elsewhere (Millar et al., 1959). The data refer to μg of free base per litre of plasma, and are uncorrected for losses in recovery up to 30 per cent.

RESULTS

Table I shows that there was no significant change in arterial adrenaline and noradrenaline concentrations 1 hour after injection of chlorpromazine, 5–10 mg/kg. When graded haemorrhage was induced (starting 75 minutes after chlorpromazine injection) circulating adrenaline levels increased progressively with each sample withdrawal. Increases in plasma noradrenaline were more moderate and occurred to a marked degree only in the later stages of haemorrhagic hypo-

TABLE I

Plasma adrenaline (A) *and noradrenaline* (N) *concentration* (μg/litre) *before and after chlorpromazine* (5–10 mg/kg), *and during haemorrhagic hypotension.*

	Chlorpromazine				Graded haemorrhage (each sample 10 ml/kg)											
	Before		After 60 min		75 min		90 min		105 min		120 min		130 min		150 min	
Study No.	A	N	A	N	A	N	A	N	A	N	A	N	A	N	A	N
1	0.2	0.1	0.0	0.1	0.5	0.0	0.7	0.1	2.2	0.1	4.6	0.2	26.5	7.1	—	—
2	0.4	0.6	0.3	0.5	2.4	0.3	2.1	0.1	2.6	0.4	2.3	1.1	3.7	1.9	11.7	5.1
3	0.4	0.2	0.5	0.5	1.8	0.1	6.0	0.4	10.5	0.8	18.4	5.2	—	—	—	—
4	0.0	0.2	0.0	0.3	0.4	0.3	0.9	0.3	2.2	0.6	4.4	1.8	21.3	10.9	—	—
5	0.0	0.0	0.2	0.2	0.3	0.3	0.5	0.2	1.0	0.5	4.1	1.4	12.9	6.2	42.3	23.6
Means	0.2	0.2	0.2	0.3	1.1	0.2	2.0	0.2	3.7	0.5	6.8	1.9	16.1	6.5	27.0	14.4
B.P.*	107	103	105	84	92	76	78	52	59	37	41	28	24	25	20	

*Mean arterial blood pressure before and after withdrawal of each blood sample.
Studies 1 and 2, chlorpromazine 5 mg/kg ; studies 3–5, chlorpromazine 10 mg/kg.

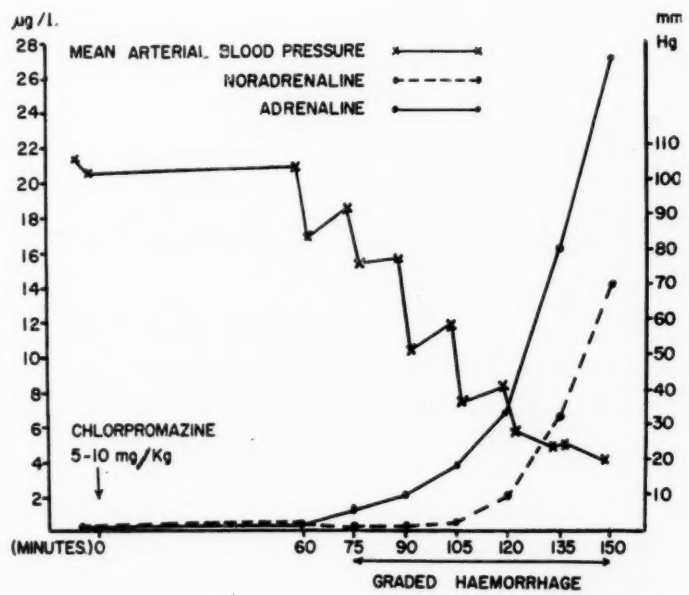

FIG. 1

Mean plasma adrenaline and noradrenaline concentrations, with average mean arterial blood pressure, in five dogs before and after intravenous chlorpromazine, and during graded haemorrhage induced subsequently.

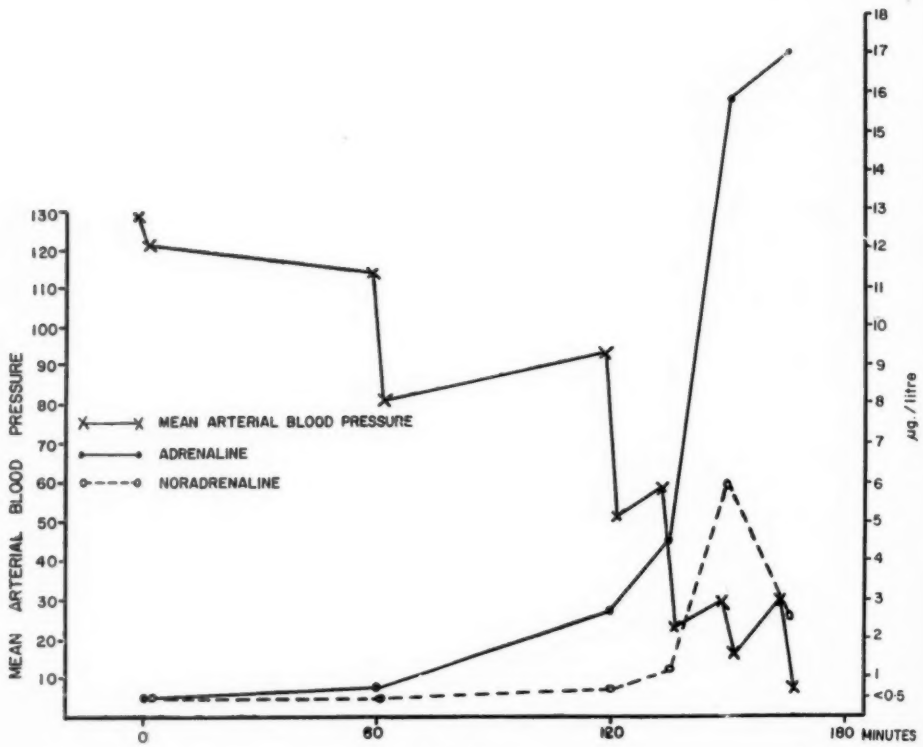

FIG. 2

Mean plasma adrenaline and noradrenaline concentrations, with average mean arterial blood pressure in eight dogs subjected to graded haemorrhagic hypotension.

tension. Figure 1 illustrates the mean values for the five studies.

The average blood volume withdrawn from the five dogs, before complete circulatory collapse occurred, was 58.4 ml/kg (range 50.7–63.6 ml/kg).

DISCUSSION AND SUMMARY

These data fail to demonstrate significant depression, by chlorpromazine, of the rise in circulating catechol amine levels which accompanies progressive haemorrhagic hypotension. This is evident from a comparison of figures 1 and 2. Figure 2 is reprinted from our previous paper (Millar and Benfey, 1958) and shows the catechol amine levels attained during haemorrhagic hypotension in dogs which have not been given chlorpromazine.

In contrast to the potent anti-adrenergic compound dibenzyline (Millar et al., 1959) chlorpromazine injection per se does not appear to alter arterial adrenaline and noradrenaline concentrations, nor does it limit the blood volume which can be withdrawn prior to circulatory failure.

It is concluded that chlorpromazine does not reduce the sensitivity of dogs to autonomic stimulation induced by circulatory stress. The central component of the sympathetic nervous system appears to be fully reactive following intravenous injection of chlorpromazine.

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ANAESTHESIA WITH HYDROXYDIONE (PRESUREN)

BY

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STEROIDS were first introduced as an anaesthetic by Murphy and his colleagues (1955) and in the published reports of several thousand administrations, which have been admirably summarized by Ditzler and Dumke (1957), many advantages have been claimed. However real these may be, the disadvantages of early preparations (Viadril) which caused a high incidence of venous thrombosis, produced anaesthesia slowly, and had to be given in the form of an intravenous drip, discouraged the widespread use of the agent. More recently 21-hydroxy-pregnane-3, 20-dione, as the water soluble sodium hemisuccinate, has become available under the name of Presuren.* It may be given as a single intravenous injection (Stedtfeld, 1957), and with a lower incidence of vein complications, allows steroids to be employed as a practical and convenient form of anaesthesia.

In an attempt to assess the value of the claims which have been made, I have used Presuren on more than 200 occasions during which particular attention was paid to the effects on respiration, blood pressure, and the immediate postoperative period.

Presuren is available as a white crystalline powder in vials containing 0.5 and 1 gram, which dissolves readily in warm, normal saline. One gram may be regarded as an average dose, but less may be necessary in the aged or the gravely ill (Opderbeke, 1957). Repeated injections may be made, but since there is a time lag before the full effect is produced, there may be difficulty in judging the dose accurately.

Concentrations of 2.5, 5, and 10 per cent have been used. Clinically there seems to be little to choose between these different concentrations, but 5 per cent proved the most convenient for general use, and was the one most commonly used in this series.

OBSERVATIONS

Induction.

Induction with Presuren is usually smooth and pleasant for the patient, with unconsciousness supervening about 4 minutes after the injection. The maximal effect appears to be reached after a further 2–3 minutes. Unfortunately, as with other steroids, Presuren is an irritant and the injection may cause pain. This is usually in the upper arm, aching in character, and often spreads along the venous distribution. It rarely occurs at the site of injection unless the solution has been deposited inadvertently outside the vein. Pain occurred in about 35 per cent of the cases, but in addition, thrombophlebitis and venous spasm were also seen (table I).

TABLE I

Vein complications in 200 *cases*

	Pain	Spasm	Thrombo-phlebitis
Number of cases	69	7	7
Incidence in cases	34.5%	3.5%	3.5%

The venous spasm resolved after about 2 hours without any residual signs or symptoms, and the thrombophlebitis responded quickly to conservative measures. Pain at the time of injection can be relieved by a variety of means, of which vigorous massage proved most effective. Injections of saline or a weak local analgesic solution may be helpful, and it has been suggested that

* Registered trade name of A. G. Schering, Berlin. Chemically this substance is identical to Viadril, but certain physical properties are different. To distinguish the form of hydroxydione used in this investigation, the trade name Presuren has been used throughout.

the latter may avoid pain and irritation altogether if it is injected at the same time as the steroid (Galley and Lerman, 1959).

Venous complications are reduced considerably if large veins only are chosen (those in the antecubital fossa being most suitable), and the injection made as fast as possible through a No. 1 hypodermic needle.* The concentration of hydroxydione seems to play no part in the incidence of these complications and the suggestion by Harbut (1957) that cold solutions cause more pain than those which are warm was not borne out in practice.

An excitement stage was not seen although movements of different parts of the body, most frequently the upper limbs, were not uncommon. Such movements were often athetoid in character, and while not necessarily related to painful stimuli they could be provoked during light anaesthesia by external stimulation (Taylor and Shearer, 1956).

Effect on Respiration.

All respiratory studies were made with a low resistance dry gas meter. This was attached to a face mask which incorporated a unidirectional expiratory valve. The volume of air inspired in 1 minute was measured, and the respiratory rate counted. Thus the estimation of tidal volume was based on an average reading.

Within 5 minutes of receiving Presuren there was an average fall in the tidal volume of about 150 ml. This represents a fall of about 30 per cent on the pre-anaesthetic value. During the next 5 minutes, a further slight fall may be expected, but thereafter little change occurs until the respiratory exchange increases again as the result of lightening anaesthesia or painful stimulation (fig. 1).

The type of premedication used (papaveretum 21.6 mg and scopolamine 0.40 mg, or pethidine 50 mg and atropine 0.65 mg, or atropine alone) had little effect on these readings, although the mechanism by which the alteration in tidal exchange was brought about differed. In those who had received the first, "heavy", premedication, a fall in minute volume was usual. In those who received the "light" premedication, a fall in minute volume might be seen, but a rise in respiratory rate was characteristic (table II), and has been already noted by Hohmann (1958).

TABLE II

The effect of premedication on minute volume and respiratory rate.

		Rise	Fall	No change
"Light" premedication (50 patients)	Rate	41	2	7
	Min. vol.	7	27	16
		Rise	Fall	No change
"Heavy" premedication (50 patients)	Rate	18	11	21
	Min. vol.	7	35	8

The average tidal volume after premedication and before anaesthesia was about 400 ml in both groups, but a few exceptionally high readings were encountered. These can be explained on the basis of emotional factors, and, as might be expected, were slightly more common in lightly premedicated patients. The administration of recognized antidotes to respiratory depression had no effect. However, nalorphine given to patients in whom depression was due primarily to premedication, was found to improve the respiratory exchange.

Presuren was administered to three patients who had previously suffered from poliomyelitis. In two, the disease had been contracted in childhood more than 20 years previously, leaving residual symptoms in the legs only. The third patient had suffered a more recent attack with extensive bulbo-spinal involvement, but with good return of respiratory function. In all three, marked respiratory depression was seen soon after the injection of Presuren, and in two this proceeded to apnoea. No adequate explanation can be offered for this dramatic occurrence, but recovery after artificial respiration was uneventful.

Case History.

Housewife, aged 35, for tonsillectomy. History of poliomyelitis when 7 years old, leaving residual weakness of left leg only, but able to lead a normal active life. Premedication, pethidine 50 mg and atropine 0.65 mg. Before induction with 750 mg Presuren the tidal exchange was 360 ml and the respiratory rate 18 per

*0.81 mm diameter = S.W.G. 21.

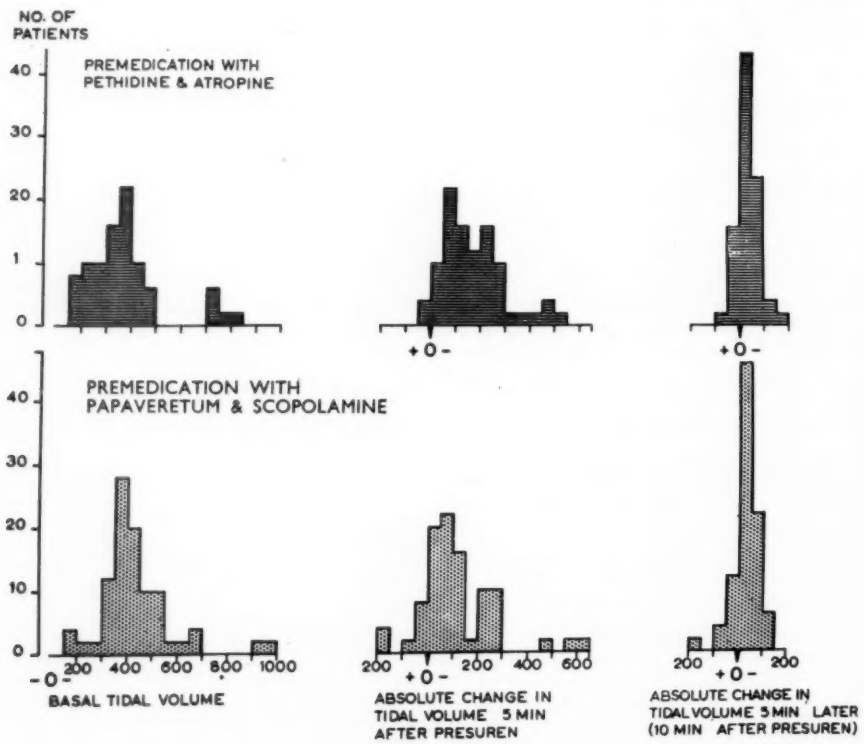
NO. OF PATIENTS
PREMEDICATION WITH PETHIDINE & ATROPINE
PREMEDICATION WITH PAPAVERETUM & SCOPOLAMINE
BASAL TIDAL VOLUME
ABSOLUTE CHANGE IN TIDAL VOLUME 5 MIN AFTER PRESUREN
ABSOLUTE CHANGE IN TIDAL VOLUME 5 MIN LATER (10 MIN AFTER PRESUREN)

FIG. 1

Histograms showing the effect on respiration of 1 gram of Presuren. The results were obtained from fifty patients who received pethidine 50 mg and atropine 0.65 mg and fifty more who received papaveretum 21.6 mg and scopolamine 0.40 mg, as premedication. The basal tidal volume was recorded in the anaesthetic room approximately 1 hour after premedication, but before the induction of anaesthesia.

minute. The patient fell asleep within 3 minutes and 2 minutes later the tidal exchange had fallen to 70 ml and the respiratory rate had increased to 28. Complete apnoea supervened and during this time the patient was intubated and artificial respiration performed with oxygen. The operation was performed and no additional drugs were needed. The apnoea lasted for 30 minutes and within 5 minutes of restarting spontaneous respiration the tidal exchange was 150 ml and the respiratory rate 24. One hour after the induction the patient had recovered consciousness and the respiratory exchange had returned to normal.

Only two other cases of prolonged apnoea during steroid anaesthesia appear to have been recorded (Howland et al., 1956; Schwarz, 1958).

Effect on the Cardiovascular System.

Five minutes after the injection of Presuren, there is usually an average fall in the blood pressure of about 20 mm Hg systolic. Thereafter there may be a slight fall of about 10 mm Hg (fig. 2). In many instances, however, there is no further reduction, and often indeed a slight rise.

The pulse rate is variable, but such changes as do occur rarely exceed a rise or fall of 10 per minute and can therefore be ignored (table III).

Neither the effects on the blood pressure nor the pulse rate appear to be affected significantly by the type of premedication received by the patient. No cardiac arrhythmias were observed.

Dilatation of superficial veins occurred soon after consciousness was lost and although this may be a typical response to the release of sympathetic tone to be expected under general anaes-

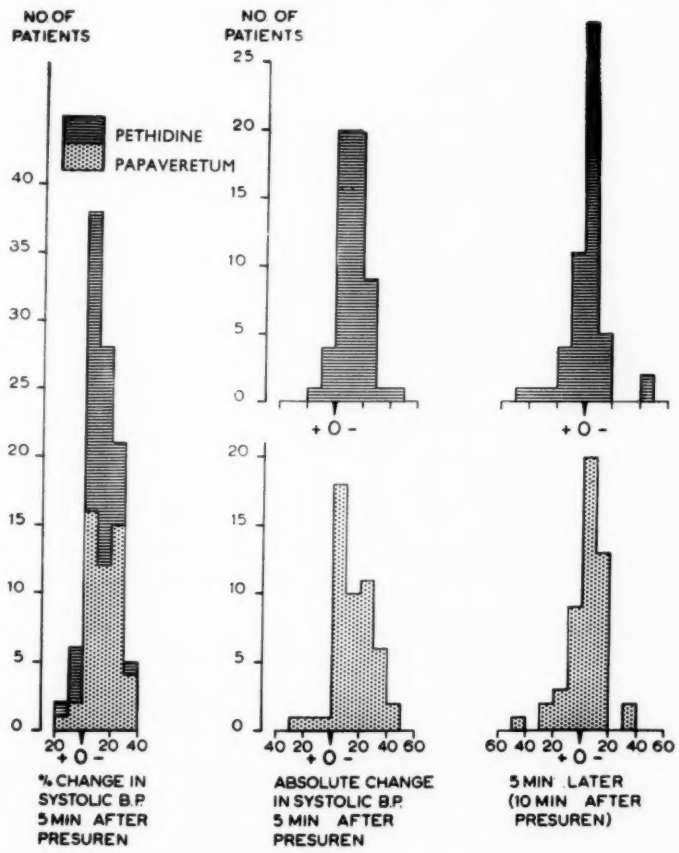
NO OF PATIENTS
PETHIDINE
PAPAVERETUM
% CHANGE IN SYSTOLIC B.P. 5 MIN AFTER PRESUREN
NO OF PATIENTS
ABSOLUTE CHANGE IN SYSTOLIC B.P. 5 MIN AFTER PRESUREN
5 MIN LATER (10 MIN AFTER PRESUREN)

FIG. 2

Histograms showing the effect of Presuren on the systolic blood pressure. 1 gram of Presuren was administered to fifty patients who received pethidine 50 mg and atropine 0.65 mg, and fifty more who received papaveretum 21.6 mg and scopolamine 0.40 mg as premedication. The differences between the two groups are small, but a slightly greater, and a slightly more prolonged fall in those who had received the heavier premedication can be noted.

thesia, it may be responsible for the small drop in blood pressure observed in most of the cases.

Other Observations.

Presuren has been used in association with a variety of other anaesthetic agents and muscle relaxants. Although adequate relaxation may be obtained using smaller doses of relaxants than usual, such an observation can only be regarded as a clinical impression. Variables such as the depth of anaesthesia and physical state of the patient are important factors in influencing the degree of muscular relaxation and it is possible that they may not have been fully taken into account in some reports (Just and Ibe, 1956).

When followed by halothane, marked respiratory depression and hypotension were observed. A return to normal levels occurred after discontinuing the halothane.

TABLE III

The effect of Presuren on pulse rate

Premedication	Rise	Fall	No change
"Light"	21	22	7
"Heavy"	24	17	9
	45	39	16
Total		100	

"No change" was recorded where the pulse rate remained within a range of ± 4 per minute. Numbers refer to patients.

Case History.

Female, aged 36, for removal of thyroid adenoma. Premedication with papaveretum and scopolamine 1 hour before induction with 1 gram Presuren. Ten minutes later, during which time no further drugs were administered, the blood pressure had fallen from 130 mm Hg systolic to 120, and the tidal exchange from 390 ml to 360 ml. Five minutes after breathing 1 per cent halothane in air, the blood pressure fell to 80 mm Hg, and the tidal exchange to 200 ml. These values returned to their normal level within a few minutes of stopping the halothane.

Nitrous oxide, trichloroethylene and ether were used after induction with Presuren uneventfully.

Laryngeal and pharyngeal reflexes tend to be depressed to such an extent that laryngoscopy is nearly always, and intubation frequently, possible without the addition of other anaesthetic drugs or muscle relaxants. Even if the patient responds to laryngeal stimulation by coughing or closure of the glottis, spasm is unusual (laryngeal spasm was provoked in one case only).

INDICATIONS

The indications for steroid anaesthesia may be difficult to define accurately. Presuren appears to be well tolerated in the aged, and although it is broken down in the liver (Jakoby and Tomkins, 1956) and excreted rapidly by the kidneys (Gardocki et al., 1956), recovery was not delayed after using the drug in patients with gross cirrhosis of the liver and hepatic failure (10 cases), and in others with a high blood urea (8 cases).

The high incidence of pain following injection may be regarded as a contra-indication to its use in young children, although Landau (1958) suggests that it is well tolerated even by the young.

A more uniform level of anaesthesia can be obtained with Presuren than with thiopentone, so that with added nitrous oxide it may prove convenient in operations requiring a relatively light level of anaesthesia. By itself, Presuren provides good sedation during operations performed under local analgesia.

Although most indications and contra-indications for the use of a drug are relative, a very real case can be made for anaesthesia with Presuren in two instances:

(1) In the chronic alcoholic with a history of being "anaesthetic resistant". In three such patients Presuren gave a smooth trouble-free induction of anaesthesia.

Case History.

Ex-guardsman, aged 68, weighing 250 lb., a known alcoholic, for dilatation of urethral stricture. On previous occasions this man had required about 1 gram of thiopentone and a muscle relaxant to allow the operation to be performed. With thiopentone alone, a violent excitement stage was usual. After premedication with papaveretum 21.6 mg and scopolamine 0.4 mg, 1 gram of Presuren produced a peaceful unconsciousness in 6 minutes. With the addition of nitrous oxide and oxygen, bouginage was performed without difficulty. The patient recovered consciousness after about 30 minutes.

(2) In laryngoscopy and bronchoscopy where it is desirable that the patient should continue breathing spontaneously during the examination. As a rule, this can be performed under 1 gram of Presuren only, but cocainization of the larynx as well is recommended.

Postoperative Course.

The impression that patients recovering from steroid anaesthesia exhibited a sense of well-being in the immediate postoperative period was confirmed (Galley and Rooms, 1956; Lerman, 1956). A state of euphoria was not unusual. The incidence of nausea and vomiting was about 10 per cent, being slightly more common in those who had received morphine for premedication than in those who received pethidine or atropine only.

Since the majority of the patients in this series received supplementary anaesthesia, it was difficult to estimate the duration of effect of Presuren by itself. Although this may be of the order of 30–40 minutes, the period of unconsciousness will be prolonged for several hours by the use of additional drugs. Had thiopentone been used in similar circumstances, a much quicker recovery would have been expected.

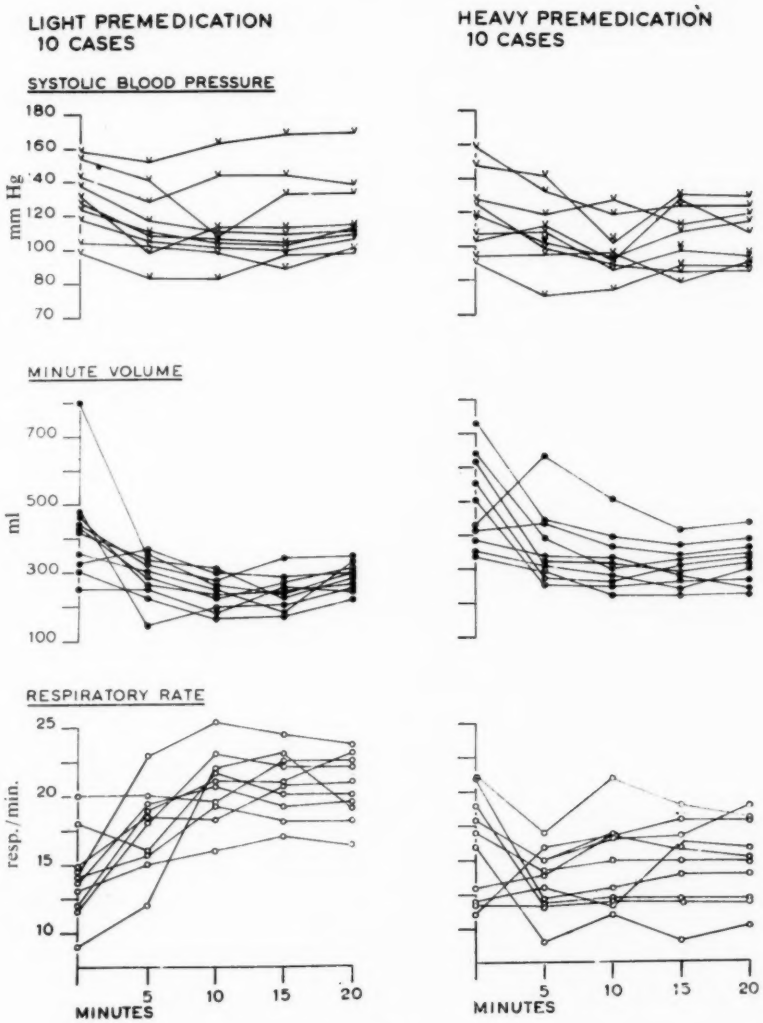

FIG. 3

Summary of ten cases who received 1 gram of Presuren after "light" premedication (pethidine 50 mg, atropine 0.65 mg) and ten cases after "heavy" premedication (papaveretum 21.6 mg, scopolamine 0.40 mg). The initial recordings were taken before induction, and further readings were made at 5-minute intervals after the injection of Presuren. No other drugs were administered during the period of observation and the patients received no operative stimulation.

(1) Systolic blood pressure. There is a slight fall in both groups which tends to be prolonged into the second 5 minutes in the "heavy" premedication group.

(2) A fall in minute volume occurs in the first 5 minutes, being most obvious in the "heavy" premedication group. After 10 minutes the recordings remain unchanged or tend to rise.

(3) While there is no obvious change of pattern in the respiratory rate in the "heavy" premedication group, in those who had received the "light" premedication, the rate rises. This rise occurs throughout the first 10 minutes after administration of Presuren.

This prolonged recovery period, which may often be due to a combination of drugs, is usually associated with depressed respiration and a low blood pressure. In these circumstances, a potentially dangerous situation arises. Six patients were encountered in this series who behaved in this manner and required careful supervision during the postoperative period.

SUMMARY

Presuren is a further stage in the development of steroid anaesthesia, with convenience, speed of action, and reduced incidence of vein complications as advantages over its predecessors.

It may cause a slight fall in systolic pressure and some respiratory depression, though rarely to a significant extent (fig. 3).

It is often difficult to judge the dose of Presuren sufficiently accurately to allow it to be used as a sole anaesthetic. The addition of premedication or supplementary anaesthesia can alter the response to this agent and tend to delay recovery.

The incidence of pain on injection and venous thrombosis cannot be ignored and contra-indicate the use of Presuren in the young. It is, however, well tolerated by the aged and the sick, and the sense of well-being in the postoperative period is impressive.

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CHOICE OF AN ANAESTHETIC FACEPIECE

BY

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THOUGH much time and attention has been devoted to the design of the anaesthetic facepiece, with regard to the requirements of simplicity of construction, ruggedness, ease of cleaning, comfort to the patient and goodness of fit, there are two other requirements which, although important, are seldom considered. These are (*a*) the mechanical deadspace which the facepiece adds to the anatomical deadspace of the patient and (*b*) the cost of the facepiece.

DEADSPACE

The average anatomical deadspace of a normal adult is approximately 150 ml. In calculating the effective alveolar ventilation this anatomical deadspace volume must be subtracted from the tidal volume. The ratio between deadspace volume and tidal volume is very important for it governs both the uptake of inspired gases and the excretion of carbon dioxide. The closer this ratio approaches to unity the less effective is the alveolar ventilation. The conditions pertaining to clinical inhalational anaesthesia tend always to reduce the volume of effective alveolar ventilation. The respiratory centre is depressed by narcotics and anaesthetics and the tidal volume is consequently reduced (the patient breathing spontaneously). The functional deadspace (anatomical + apparatus deadspace) is invariably increased by the apparatus used.

In any apparatus involving its use, the facepiece contributes the major proportion of this increased deadspace, assuming flows of fresh gases are used which are adequate to eliminate rebreathing from the corrugated tubing (Molyneux and Pask, 1951). With the oxygen-rich gas mixtures currently used in clinical anaesthetic practice normal oxygenation may be maintained with subnormal volumes of effective alveolar ventilation; but the excretion of carbon dioxide, which is critically dependent on the volume of effective alveolar ventilation, will be decreased, and thus the carbon dioxide content of the blood will rise. The clinical signs of this, certainly in the early stages, may be masked by anaesthesia. It is thus evident that the deadspace of the facepiece is a very important factor in inhalational anaesthesia, and especially so in systems, such as the Magill attachment, where carbon dioxide elimination depends on volume displacement, and in subjects such as children whose tidal volumes are small and approach the functional deadspace.

It is rather surprising that so little effort appears to have been made, up to the present, to measure the mechanical deadspace of anaesthetic facepieces. Gillespie (1948) suggested that the approximate content of most anaesthetic masks was 150 ml. Stephen (1952) reported that a child's facepiece has a capacity of 250 ml and drew attention to the possibility of rebreathing. Schwartz et al. (1957) emphasized that an adult's medium-sized anaesthetic mask has a volume of 120 ml and a child's mask has a volume of 40 ml. Leigh and Bolton (1948) have pointed out that in children and infants the mask and equipment doubles or trebles the deadspace. Vale (1958) re-emphasizes this important fact by drawing attention to the capacity of an infant's anaesthetic mask and also to the measurements of anatomical deadspace in children reported by Hall (1955). All these workers were content to measure or calculate the capacity of the facepiece itself, although it is obvious that when fitted to the face of a subject some of that capacity will be occupied by the subject's physiognomy. This has been taken into account by Clarke (1958) who fitted under standard conditions a single no. 4 McKesson facepiece to 25 male and 20 female cadavers, and measured the deadspace with the mask in position. He found that the mechanical deadspace

of the facepiece varied between 76 and 107 ml for the males and between 87 and 110 ml for the females. To these figures he added 28–30 ml for the mechanical deadspace of the angled connector and expiratory valve usually found on the Magill attachment.

METHOD

The same measurements as described by Clarke (1958) were carried out. The terminology "mechanical deadspace" is preferred to that of "potential deadspace" for the space measured. The use of the latter terminology by Clarke is based on the possibility of channelling within the mask in the same way as occurs within the bronchi, but until this is proven it should be assumed that the whole of the "mechanical deadspace" is operative with each respiration. The mechanical deadspace of nine adult size anaesthetic facepieces when applied to the faces of 21 adult subjects with full dentition—11 males and 10 females aged between 25 and 35 years—was measured, using a modification of a method suggested by Woolmer (1955). The nine masks comprised seven different types. These are illustrated in figure 1.

The volume measured was that of the mask,

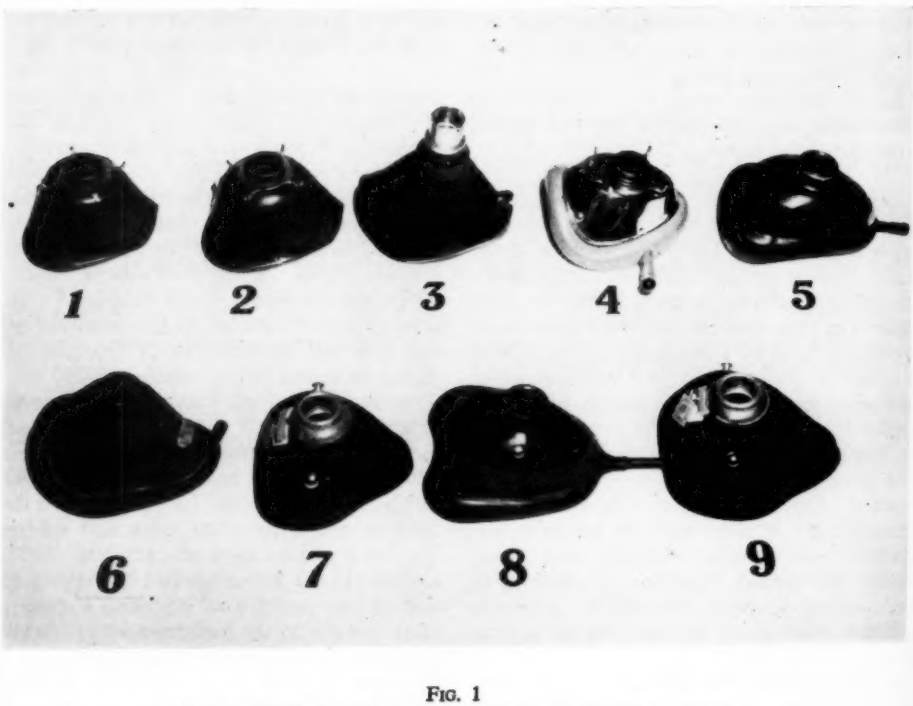

FIG. 1

The anaesthetic facepieces used in the measurements reported.

1. Draeger, Foregger type, size 3, M.9503.
2. Draeger, Foregger type, size 4, M.9504.
3. Foregger, Connell type, adult size, MCS 22.
4. Draeger, Draeger type, chrome base with detachable pad, size 4, M8155 and M6202.
5. Franklin, Rubber Co., London; anaesthetic facepiece with detachable inflatable rubber pad, size 4.
6. B.O.C. Connell type, size 5, MS207.
7. M.I.E. Everseal anaesthetic facepiece, size 3.
8. B.O.C. Anaesthetic facepiece body with detachable inflatable pad, size 5, 22/502 and 22/506.
9. M.I.E. Everseal anaesthetic facepiece, size 4.

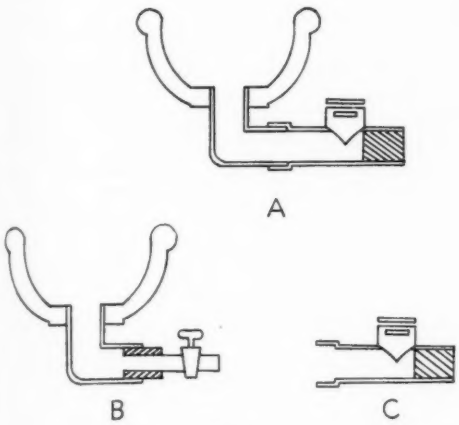

FIG. 2
Diagram of the mask, angle piece and valve piece assembly.

angle piece and valve piece up to the expiratory valve of a standard Magill assembly (fig. 2A).

The mask to be tested was assembled with a standard angle piece. The angle piece was occluded by a cork at the point to which the valve piece of the Magill assembly would overlap. Through this cork a glass tube with burette tap was led. The mask and angle piece as illustrated in figure 2B were now completely submerged in water in a large basin, the burette tap being open. When all the air was displaced from the system the subject, holding his breath, lowered his face into the water and fitted the mask firmly to his submerged face, excess water being displaced through the burette tap. The tap was then closed and the subject removed the mask assembly, still fitted to his face, from the water. Holding the mask horizontally, the subject now gently disengaged his face from the mask, taking care not to spill any water. The water remaining in the facepiece was now carefully decanted through the burette tap into a measuring cylinder. The mean of two readings per mask per subject was recorded. The volume of the valve piece—the proximal end occluded by plasticine up to the expiratory valve (fig. 2C)—was measured separately; this volume then being added to the mean individual mask volume. The volume of the glass tube and tap assembly was subtracted from this

TABLE I

Volume of mask deadspace in ml—individual readings.

Mask	1	2	3	4	5	6	7	8	9
Males									
G.R. 165 lb.	95	100	145	147	150	167	152	212	190
W.L. 165 lb.	90	95	157	162	170	174	190	257	270
H.duT. 172 lb.	81	89	155	142	159	157	185	257	235
P.H. 163 lb.	82	87	125	140	135	115	142	177	205
T.V. 155 lb.	80	89	144	140	155	165	165	207	192
B.S. 150 lb.	62	77	127	132	135	155	142	185	195
D.J. 135 lb.	77	95	152	132	160	167	155	197	217
L.B. 150 lb.	62	77	115	140	135	160	135	180	167
A.B. 150 lb.	70	77	130	125	125	150	155	165	185
G.H. 176 lb.	74	86	148	121	135	105	156	162	175
J.O. 149 lb.	81	86	101	124	134	94	166	187	203
Mean	78	87	136	137	145	146	158	198	203
S.D.	10	7	17	11	14	27	17	31	28
Females									
V.D.M. 135 lb.	88	106	146	143	131	161	138	188	218
D.P. 115 lb.	86	88	146	148	148	161	178	211	248
J.M. 115 lb.	93	108	151	163	161	161	188	148	246
M.G. 130 lb.	68	73	108	133	131	153	148	158	173
V.B. 116 lb.	86	91	136	148	141	168	161	203	166
E.W. 126 lb.	71	86	138	141	133	151	138	186	166
R.B. 137 lb.	86	91	146	126	148	166	176	211	226
V.D.M. 110 lb.	73	91	133	141	136	166	136	183	146
S.V.H. 130 lb.	71	86	136	136	123	156	148	181	173
M.H. 98 lb.	81	91	138	136	133	161	143	173	186
Mean	80	91	137	141	138	160	155	184	194
S.D.	8	10	11	10	11	5	18	20	35
Overall Mean	78	89	137	139	141	153	157	191	198
S.D.	9	9	15	11	13	21	17	27	32

total to give the volume of the complete assembly (fig. 2A).

RESULTS

The results of measuring the mechanical deadspace and the cost analysis are set out in tables I and II. The deadspace of the nine masks tested ranged from 62 to 270 ml with the mean value showing a range of from 78 (S.D. = 9) to 198 (S.D. = 32) ml.

The tables are arranged for convenience so that they read from least to greatest from left to right in terms of mean values. The ratio that these volumes bear to that of the anatomical deadspace —taken as 150 ml—is also appended. Note that masks 1 and 2 are different adult sizes of the same type of mask, as are masks 7 and 9.

DISCUSSION

It will be seen from table II that of the masks tested, in all but one type—of which there were two sizes—the mechanical deadspace observed ranged from just less than to considerably more than the anatomical deadspace. This additional deadspace will lead to a fall in the volume of effective alveolar ventilation and consequently to inefficient elimination of CO_2. It must therefore contribute materially to the respiratory acidosis that often accompanies inhalational anaesthesia (Cullen, 1954; Watrous et al., 1950; Dripps and Severinghaus, 1955). In choosing an anaesthetic facepiece, one with a low deadspace volume should ideally be chosen.

Though all the subjects tested show a wide range of deadspace volumes over the nine masks tested (table I), only two sets of measurements consistently parallel the mean figures in progression from lowest volume to greatest. This is no doubt due to variation in facial features. We regret that we have no satisfactory method of typing faces by measurement of their parameters, though the type of face is undoubtedly of importance in determining the volume of mechanical deadspace in any particular mask, e.g. a small angular face will fit right into a large mask, leaving much less deadspace than when the same mask is fitted to a large flat or concave type of face.

Cost is another factor that must be considered in choosing an anaesthetic facepiece. This is especially so when equipping a large hospital. The costs of the facepieces tested ranged from that of the cheapest up to nearly seven times that price (table II).

In the work here reported we were able to take advantage of a request to test several types of anaesthetic facepieces—adult sizes only were supplied—with a view to recommending one that should be adopted as a standard item of equipment for an extensive hospital system. Since actual local costs are of no value to others, we have introduced them here by taking the lowest price of a mask as unity and expressing the prices of the other masks as multiples of this cost unit.

It must be remembered that the final choice should also take into consideration the other requirements mentioned earlier, viz. simplicity, ruggedness, ease of cleaning, comfort to the patient and goodness of fit. In this respect it is of interest to note that our own tests of these masks included daily clinical use for a period of three months in a very busy general hospital. In this period one of the most expensive facepieces became worn and finally torn at the point of contact with the nasal bridge.

TABLE II

General table of results.

Mask	1	2	3	4	5	6	7	8	9
Deadspace overall mean ml ...	78	89	137	139	141	153	157	191	198
S.D.	9	9	15	11	13	21	17	27	32
Mask deadspace/*anatomical deadspace	0.5	0.6	0.9	0.9	0.9	1.0	1.0	1.3	1.3
Cost factor	6.52	6.9	—	5.2	1.0	3.46	4.64	2.06	4.64

*Taken as 150 ml.

SUMMARY

Observations on the mechanical deadspace of seven types of mask assembled with angle piece and valve stock, reveal that in all but one of these this volume is approximately equal to or greater than the anatomical deadspace volume of an average adult. Patients anaesthetized with such masks might be expected to experience difficulty in the adequate elimination of carbon dioxide. This should be borne in mind when choosing an anaesthetic facepiece.

It is also noted that the costs of facepieces vary greatly from each other.

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A DOUBLE-LUMEN ENDOBRONCHIAL TUBE

BY

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ENDOBRONCHIAL intubation is a well-established practice in anaesthesia for thoracic surgery, and since the introduction of the orginal technique described by Gale and Waters (1932), many ingenious tubes and blockers have been devised. These have had two objects in mind: either to render existing methods more efficient (Stuetzbecher, 1953; Vellacott, 1954; Green and Gordon, 1957) or to simplify, and thus place a method at the disposal of those who make no claim to being expert thoracic anaesthetists (Macintosh and Leatherdale, 1955).

These technical advances do not, however, overcome the principal disadvantage of one-lung anaesthesia—the shunt of blood through a non-aerating lung. Although the function of the lower lung may be increased by the gravitational flow of blood in the lateral position (Rothstein, Landis, and Narodick, 1950), this may be inadequate compensation for many patients. It is in such circumstances that the double lumen catheter may prove its worth. Independent control of the lungs not only provides the safeguards offered by simple endobronchial intubation, but at the same time permits a more efficient ventilation to be carried out. Moreover, the movement and degree of distension of the lung on the side of operation can be adjusted to suit the surgical needs in a way that would be impossible with endotracheal intubation and with the use of endobronchial blockers. The Carlens tube, which has these advantages, was described in 1949 and its use in anaesthesia and bronchospirometry has been recorded in a number of instances (Bjork, Carlens and Friberg, 1953; Middleton, 1957). Such a tube has the added attraction of being made so that its shape conforms to the angle made by the left bronchus to the trachea, a feature emphasized also in the construction of the Macintosh-Leatherdale tube and blocker. In this way the stability of the tube is increased and the risk of displacement of the terminal cuff reduced to a minimum.

The tube described here is a modification of the Carlens catheter, the principles of which are retained, but there is no carinal hook, as this often causes considerable difficulty in the introduction of the tube and is probably unnecessary. It is made in a manner similar to that of the Macintosh-Leatherdale tube, being formed by single extrusion process which has the merits of cheapness and a smooth uniform surface of compounded rubber which is more durable than a moulded tube over-dipped in latex.

The tube has a double lumen for the greater part of its length, but it continues for $2\frac{3}{4}$ inches (7 cm) as a single tube which is curved both posteriorly and to the left. This section bears a cuff at the distal end which when inflated lies in the left main stem bronchus proximal to the upper lobe orifice. The other channel of the catheter terminates just above the carina, and an airtight fit is made in the trachea by means of a second cuff (fig. 1).

The proximal end of the catheter has an anterior curve so that it lies easily in the oropharynx. It terminates as two separate tubes into which connectors may be fitted. These tubes are of equal size and are designated by the diameter of each tube in millimetres. Three differently sized tubes are made, the smallest being 6 mm in diameter (roughly equivalent to two Magill No. 4 tubes), the next 6.5 mm and the largest 7 mm.

The resistance of these catheters is dependent on their diameter and length, but the difference in resistance between the two channels of each is negligible. For comparison with an endotracheal tube, the resistance in a 7 mm double lumen catheter (both channels simultaneously) is about the same as in that of a No. 10 Magill tube of equivalent length (about 36 cm) at continuous flow rates up to 40 litres per minute.

The length of the double-lumen catheter is slightly greater than in the Macintosh-Leather-

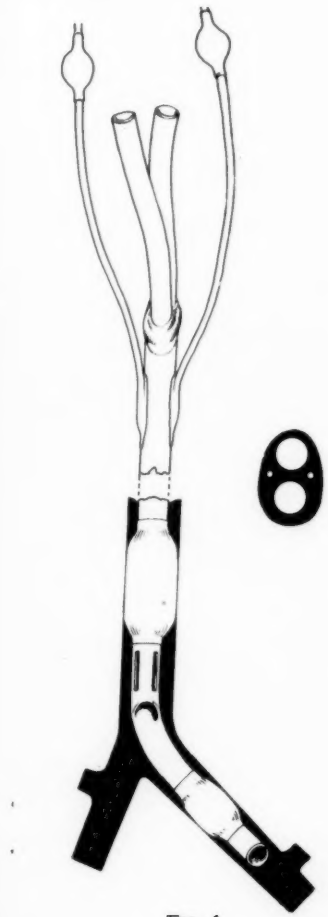

FIG. 1

The double-lumen tube in position, and cross-section of the tube above the level of the tracheal cuff.

dale tube, and this extra length has been based on bronchoscopic measurements (carina to lower teeth), observation of the position of the endobronchial section after opening the bronchus during right pneumonectomy, and by radiography with the cuffs distended with contrast medium (Dionosil).

METHOD OF INSERTION

As with the Macintosh-Leatherdale tube, the double-lumen catheter is inserted through the cords under direct vision. The laryngoscope is then discarded and the catheter advanced blindly. Because of its shape, the terminal portion will tend to enter the left main stem bronchus and will be halted when the double-lumen section reaches the carina. The bronchial and tracheal cuffs may now be inflated and the correct siting of the tube confirmed by auscultation.

On two occasions, the tube has proved life-saving by preventing soiling of the left lung with gross haemorrhage. On the first occasion the tube was introduced to provide urgent independent control of the lungs in a patient with a ruptured right lower bronchus. On the second occasion it had been used fortuitously for a right pneumonectomy for carcinoma of bronchus. After the bronchus had been divided and left open, the pulmonary artery tore, causing a haemorrhage of about 5 pints.

This double-lumen catheter is particularly suitable for all operations on the right lung and for left lobectomy. To satisfy the requirements of left pneumonectomy, a "right-sided" tube with a Green-Gordon type of cuff on its endobronchial section is undergoing trials.

ACKNOWLEDGMENTS

I am grateful to Mr. Richard Salt of this department for his help in designing the tube, and to the Leyland and Birmingham Rubber Company for their wholehearted co-operation. The tubes may be obtained from Messrs. Medical and Industrial Equipment Ltd., 10/12 New Cavendish Street, London, W.1.

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AN IMPROVISED ETHYL CHLORIDE ATTACHMENT

BY

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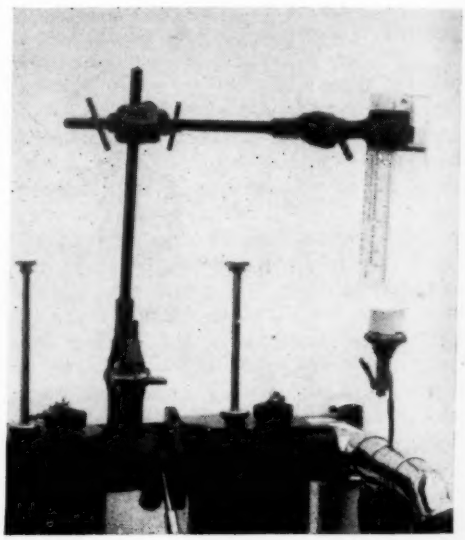
THE sequence nitrous oxide–oxygen–ethyl chloride is one which has been successfully used by a number of anaesthetists for many years, especially when a pleasant induction followed by brief but deep anaesthesia and rapid recovery, is desired—e.g. for guillotine tonsillectomy in children.

The accompanying photograph illustrates an improvisation to a standard Boyle machine with a Magill attachment which facilitates this technique. A detailed discussion of the technique will be found elsewhere (Campbell and Smith, 1955).



The tap of the reservoir-bag is placed in the vertical position and unscrewed. Through the hole thus exposed some 12 to 15 inches of suitably fine rubber tubing are threaded, and so introduced into the adjacent part of the breathing-tube. Into the end of the tubing which now remains protruding from the hole is inserted the nozzle of an ethyl chloride spray. This is in turn fixed vertically above the hole by means of an arrangement of clamps of the type used with a laboratory burette-stand. The jaws of the clamp which holds the spray can be padded with adhe sive sponge rubber to minimize the risk of breakage.

At first sight it might seem more obvious to introduce the ethyl chloride directly into the reservoir-bag, and indeed this was done for a number of years—until the introduction of antistatic rubber. The first few reservoir-bags of this material subjected to ethyl chloride perished and disintegrated after about five or six administrations; hence the reason for diverting the ethyl chloride into the breathing-tube, which is much thicker and does not deteriorate noticeably when so used. It is probable in any case that the ethyl chloride is more quickly and efficiently vaporized from the tube than from the bag.

ACKNOWLEDGMENT

I am indebted to Dr. J. S. Faulds for the photograph.

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FROM THE MORBIDITY CONFERENCE

A REPORT ON TWO CASES OF CARDIAC ARREST

BY

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CASE 1. *Circulatory Collapse following I.P.P.R.*

This incident occurred at the Royal Liverpool Children's Hospital. The patient was a mature 14-year-old girl, weighing approximately 8 stone (50 kg).

She was admitted on February 26, 1958, with severe burns sustained when her nightdress caught alight. Forty per cent of her body surface area was affected, half of this being third-degree burns. Plasma and later whole blood were transfused, and dressings were applied under morphine sedation. She passed through a critical 36 hours, showing some signs of peripheral circulatory failure, but then began to improve. The relevant biochemical investigations were carried out, and normal levels were achieved by oral and intravenous replacement therapy. On March 12 she came to theatre for the first time for skin grafting and redressing of the burns.

Premedication consisted of quinalbarbitone 120 mg 1½ hours before operation, and then morphine 10 mg and atropine 0.6 mg 1 hour before operation. A test dose of d-tubocurarine 4 mg was given and anaesthesia was induced with thiopentone 200 mg. A further 20 mg of d-tubocurarine was given. Intubation was carried out with a plain No. 8 endotracheal tube and a mouth pack was inserted. Anaesthesia was maintained in a closed circuit using 3 l. nitrous oxide and 1 l. oxygen per minute, with carbon dioxide absorption being effected in a Water's canister (adult type). Controlled respiration was maintained throughout.

During the course of the operation which lasted 2 hours, the patient was transfused with 2 pints (1 l.) of whole blood and given two supplementary doses each of 3 mg of d-tubocurarine. When the anaesthesia was discontinued, the curarization was terminated by atropine 0.8 mg followed some minutes later by neostigmine 4 mg. The patient was fully conscious when she left the theatre.

On March 18 a similar operation was carried out, the same anaesthetic being used.

On March 31 a third grafting and redressing was planned. The same premedication was given, the morphine and atropine being administered 75 minutes before operation. Since admission, the patient's condition had remained fair, apart from bouts of fever with accompanying tachycardia resulting from sepsis in the ungrafted areas. The pre-operative estimation of electrolytes was normal and the child did not look toxic.

Anaesthesia was again induced with thiopentone 200 mg, but 40 mg of suxamethonium was now used for intubation since it was planned to keep the patient on spontaneous respiration with a mixture of nitrous oxide, oxygen and trichloroethylene. Intubation, preceded by spraying of the larynx with lignocaine 2 per cent presented no difficulty and no hypoxia occurred. Approximately 1.5 ml (i.e. 30 mg) of lignocaine were used.

Controlled respiration by pulmonary inflation with a mixture of 6 l. of nitrous oxide and 2 l. of oxygen only was commenced, since spontaneous respiration had not yet begun. No trichloroethylene was at any time added. At this point the patient's colour, which until that moment had been a "healthy pink", suddenly became a deathly white. The pulse was impalpable. The pupils were dilated. No heart beat could be heard on direct auscultation of the chest wall. At 4.32 p.m. the surgeon was informed that cardiac arrest had

occurred, and the patient was immediately wheeled into the theatre on a trolley. The sequence of events was then as follows:

P.M.

4.32. Head of the trolley lowered and inflation with pure oxygen commenced.

4.33. No response. Surgeon requested to open chest and start cardiac massage. Inflation with oxygen continued.

4.34. Surgeon's hand within the thorax, and the heart massaged against the sternum.

4.38. Pericardium opened after chest wall retraction and bimanual massage instituted. Ventricles not fibrillating but no return of beat.

4.39. Adrenaline 6 ml of 1 : 3,000 solution injected into the left ventricle and massage continued. Within seconds, three forceful contractions occurred, followed by arrest and ventricular fibrillation. Whilst the defibrillator apparatus was being set up, massage was continued. It was noted that the patient's colour was now a little better, the pupils were less dilated and the carotid pulses could be palpated with each massaging stroke.

4.42. Delay due to technical hitch with defibrillator apparatus. Calcium chloride 5 ml of 10 per cent solution injected into left ventricle. No visible effect.

4.48. Defibrillator apparatus ready. Cardiac muscle tone now poor. A further injection of adrenaline 6 ml given into the left ventricle and massage continued. Two contractions, arrest and coarse fibrillation followed. Electrodes applied and three shocks delivered successively, each of 110 volts for 0.2 seconds. After several more massaging strokes, the heart started beating spontaneously at 4.51 p.m.

5.00. B.P. 70/50 mm Hg. An infusion of intravenous noradrenaline 2 mg in 540 ml of dextrose 5 per cent solution commenced. Marked improvement of force of heart beat, but severe slowing of rate noted. Intravenous atropine 0.32 mg given, with subsequent return to normal rate.

5.15. Spontaneous respiration returned. Controlled respiration instituted with suxamethonium in intermittent doses, and nitrous oxide introduced to maintain anaesthesia.

5.20. Electrocardiographic tracings from limb leads only showed normal rhythm but marked depression of the ST segment, which improved markedly during the succeeding 20 minutes.

5.45. Chest closed with waterseal drainage. Noradrenaline discontinued and blood transfusion commenced. Blood pressure maintained at 100/70 mm Hg.

7.00. Hypothermia considered to counter possible cerebral oedema, but not undertaken since the pupils were now normal in size.

7.15. Patient returned to the ward.

8.15. Consciousness returned. Patient recognized people and complained of feeling tired.

Next day. Patient felt well and a chest radiograph revealed no signs of postoperative complication. Convalescence was uneventful, and no cerebral changes have since been detected.

Two subsequent operations, on April 8 and April 25, for further skin grafts, using the thiopentone d-tubocurarine technique previously described, were uneventful.

In retrospect, it would appear that the cardiac arrest in this case was due to a combination of factors.

The blood volume of the patient was probably subnormal due to the constant slight plasma loss from the old burned areas, and the injection of thiopentone caused a very severe fall in blood pressure. The final insult was the introduction of positive pressure ventilation impeding the already reduced venous return.

Anoxia was avoided at all times during induction and the small amount of lignocaine used in spraying the larynx was unlikely to have any general effects. Relaxation was complete during easy intubation and reflex cardiac arrest was most improbable.

Immediate diagnosis of the cardiac arrest followed by rapid efficient cardiac massage were all important in the successful management of the case described.

CASE 2. *Striking Effect of Intracardiac Adrenaline.*

This incident occurred in the treatment of a case of barbiturate poisoning.

On July 10, 1958, a 65-year-old woman was admitted to the casualty department of the Liverpool Royal Infirmary. According to her husband she had swallowed an overdose of sleeping tablets. On admission she was unconscious and respirations were slightly depressed. When the casualty officer attempted to pass a stomach tube, her respirations ceased altogether. Oxygen was immediately administered through a Boyle's machine, but her pulse became imperceptible and no heart sounds could be heard. The casualty officer thereupon injected adrenaline 2 ml of 1 : 1,000 solution through the chest wall into the left ventricle, and the heart restarted.

At this stage, the patient's colour was fair and her blood pressure reading 260/120 mm Hg. While an endotracheal tube and a Water's circuit and canister were being procured, inflation was maintained with oxygen and nitrous oxide at a high flow rate and an intravenous drip of Noradrenaline was prepared. Whilst this was

being done, the heart again stopped beating. A further intracardiac injection of adrenaline was given and the heart again restarted. The endotracheal tube was now passed and inflation continued, but the heart again stopped and it was noticed that the noradrenaline was running into the tissue. The drip was discontinued and adrenaline injected into the heart for the third time. Again the heart started. The noradrenaline drip was now established satisfactorily and the blood pressure level maintained at about 100/70 mm Hg by varying the rate of flow of noradrenaline. By controlled hyperventilation it was hoped to washout any excess carbon dioxide which had been accumulated. During this phase the pupils, which had been widely dilated, started contracting.

Gastric lavage was now performed and numerous capsule remnants washed out. Bemegride 50 mg and amiphenazole 30 mg were injected alternately at intervals through the noradrenaline drip until the patient began breathing spontaneously about 45 minutes later. A total of bemegride 1 gram and amiphenazole 600 mg was given. By that time she had been transferred to the operating theatre and the intermittent positive pressure respiration continued with the Aintree respirator. Since it appeared that the blood pressure level could be maintained only by vasopressor drugs, it was considered desirable to introduce a longer acting drug and 10 mg of methoxamine was injected intravenously and continued so that it was possible to wean the patient off noradrenaline within a few hours.

In order to minimize the possible brain damage resulting from the anoxic episodes occurring during cardiac arrest, two injections of 50 per cent sucrose (20 ml) were injected intravenously to counter any cerebral oedema. As a further precaution, reduction of the cerebral metabolism by means of hypothermia was considered worth while. The difficulty was, of course, that the noradrenaline and methoxamine had produced peripheral vasoconstriction, which would hamper the process of cooling and markedly increase the chance of local frostbite. Hypothermia might also delay the excretion rate of barbiturate from the body, but both these points were considered less important than overcoming the brain damage by reducing the cerebral metabolism. A large dose of chlorpromazine might produce peripheral vasodilatation, but only at the expense of a fall in the still labile blood pressure. Eventually a dose of 25 mg was given.

Hypothermia was obtained by the surface method of cooling and, after a slow fall, a final temperature of 85°F (29.5°C) was obtained.

The patient was returned to a side ward, the cuffed endotracheal and stomach tubes being left in situ. Careful changing of the position in which the patient was lying, tracheobronchial, pharyngeal and stomach aspirations were carried out until consciousness returned. Antibiotics, intravenous fluids and vasopressor agents were given as required, the last being controlled by frequent blood pressure estimation.

After 10 hours the temperature was gradually allowed to rise and the laryngeal reflex returned. By this time, vasopressor drugs were no longer required, the blood pressure being stable, and 36 hours after admission the patient was conscious, recognizing her relatives.

Subsequently, electrocardiographs showed no evidence of myocardial damage. Slight surgical emphysema developed on the left side of the chest wall and radiography showed an underlying partial pneumothorax which cleared up quickly.

According to the patient's relatives, the episode caused no deterioration in her mental powers. She was a manic-depressive who had been treated with electroconvulsive therapy, and had attempted suicide on two previous occasions. It is not known how many tablets the patient consumed on this occasion.

In the above case the cause of the cardiac arrest was central depression due to barbiturate overdosage, causing respiratory failure followed by cardiovascular collapse. Intracardiac injections of adrenaline were successful here, but had the first injection failed, thoracotomy and cardiac massage would have been performed immediately.

With the resumption of spontaneous respiration, treatment was aimed at maintaining body functions as near normal as possible during the period of deep anaesthesia by avoiding or correcting shock, hypoxia, respiratory obstruction and peripheral circulatory collapse. So with the excretion of the barbiturates the coma lightened, during which phase the patient was nursed carefully. Hypothermia was induced for reasons already stated.

CORRESPONDENCE

DELAYED RECOVERY FROM ANAESTHESIA DUE TO UNSUSPECTED HYPOGLYCAEMIA

Sir,—It is well known that hypoglycaemia must be suspected whenever there is a delay in the return of consciousness following an operation under general anaesthesia in a known diabetic.

The following is a case of delayed recovery of consciousness due to unsuspected hypoglycaemia in a nondiabetic patient, successfully treated with intravenous glucose.

A young male aged 12 was brought to hospital with a marked paraphimosis of about 10 hours duration. Having been assured that he had had no food or drink for 5 hours, an anaesthetic was administered and the paraphimosis was reduced.

Premedication was atropine sulphate 1/100 grain half an hour prior to operation. The anaesthetic was: induction with thiopentone sodium 100 mg, and maintenance with nitrous oxide, oxygen and halothane, on a semi-open circuit. The duration of anaesthesia and operation was about 6 minutes. The child did not recover consciousness for about 4 hours, the normal recovery time for similar cases usually being between 10 and 15 minutes.

This case is interesting, as one did not suspect hypoglycaemia at first. The child was a congenitally defective spastic and was underdeveloped. We were also informed that on previous occasions he had had screaming fits followed by long periods of somnolence. He had not slept well the previous night and on one occasion he had slept continuously for 24 hours with no ill effects.

All this information was rather misleading. Assuming that the long period of sleep was a natural phenomenon in his case, we allowed the child to remain asleep, and waited for a natural recovery. As there was no sign of waking even after 4 hours, we thought of some more energetic form of treatment. An intravenous injection of 50 per cent glucose was given. After about 10 ml was given reflex movements occurred. A further 10 ml was given and at the conclusion, the child opened his eyes and tried to sit up.

On further questioning, the parents stated that the child had had no food or drink since 5 p.m. on the previous evening, so the period of preoperative starvation was about 28 hours, and not 5 hours as was thought at first.

The interesting point about this case was that hypoglycaemia occurred in a nondiabetic child due to prolonged starvation. The general state of the child together with his abnormalities in development and in behaviour, and the history as obtained at the time, had rather misled us regarding the correct line of treatment. It would have been better if diagnosis had been made and treatment started earlier, in order to prevent any further brain damage in an already handicapped child.

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